Cerebral Aneurysms

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I. Introduction

Thesis: There are different classifications of aneurysms based on their shape, formation, and size. The main form of aneurysm found in the brain is called a saccular aneurysm. The correct course of treatment for the patient will be determined by the location, size, and type of aneurysm.

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Objectives

At the conclusion of this paper, the reader should be able to:

- Identify the different types of aneurysms
- Recognize the signs, symptoms, and risk factors of cerebral aneurysms
- Understand the different ways an aneurysm can be diagnosed
- Describe the different types of treatment options available for patients with cerebral aneurysms

Cerebral Aneurysms

"The worst headache of my life." This is a common phrase used by patients who are suffering from a ruptured cerebral aneurysm. A cerebral aneurysm(s) (CA), or a brain aneurysm, is defined as "...a bulging, weakened area in the wall of a blood vessel in the brain, resulting in an abnormal widening or ballooning greater than 50% of the normal diameter (width)."[1] CAs can occur in arteries located anywhere in the brain with the most common place being the anterior portion of the brain where oxygen rich blood is deposited.[1] CAs are less commonly found in veins than in arteries.[2] There are different classifications of aneurysms based on their shape, formation, and size. The main form of aneurysm found in the brain is called a saccular aneurysm. The correct course of treatment for the patient will be determined by the location, size, and type of aneurysm.

Background

A normal artery has three muscular layers that form its arterial wall; tunica intima, tunica media, and tunica adventitia. If an artery has an abnormality and consists of only two muscular layers instead of three, an aneurysm is more likely to form due to its weakened wall.[2] Aneurysms are also thought to be caused by "... a multifactorial process that leads to degenerative changes in the blood vessel wall. The degenerative changes could be due to collagen deficiency or congenital weakness of the arterial walls."[3] Even though aneurysms are formed from the weakening of arterial or venous walls, it takes many years for them to grow and develop. Overtime, the pounding of the blood through the weakened spot in the vessel's wall expands into a sac, causing an aneurysm.[1]

It is approximated that 3-5% of Americans have a CA with around 30,000 Americans suffering from a ruptured CA each year.[5] CAs are more commonly found in adults between the age of 30-60 years and are more commonly found in women than men, especially if they are over the age of 50.[3] CAs very rarely are found in children as it usually takes a long period of time for an aneurysm to form.[3] If a person has one known aneurysm, there is a 20-30% chance for that person to have additional aneurysms.[3] If an individual were to have more than one aneurysm, the additional aneurysm will often be located on the opposite side of the brain from where the first aneurysm was discovered.[1]

One of the ideal places for aneurysms to form is at forks or branches (bifurcations or junctions) in the arteries. This is due to the extra stress being forced on the vessel when pumping blood throughout the brain. The most common junctions where CAs are found include: at the internal carotid artery and the posterior communicating artery or at the bifurcation, the middle cerebral artery, the middle cerebral artery bifurcation, and approximately 85% being located in the anterior circulation of the Circle of Willis (COW)[3] The COW, depicted in Figure 1, is made up of two anterior, a middle, and two posterior cerebral arteries, two anterior and two posterior communicating arteries.[4] Two vertebral arteries join together to form the basilar artery which then leads into the COW.[4] The most common junctions for aneurysms to occur in the posterior arteries of the brain are the basilar and superior (anterior) inferior cerebellar arteries and the vertebral and posterior inferior cerebellar arteries.[3]

The arteries in the brain have a high pressure of blood flow compared to the pressure found in the veins, which bring deoxygenated blood back to the heart.[6] If there is a weakening in the artery wall, the high pressure creates the bulging and potential rupture of the aneurysm. When an aneurysm starts to form a bulge or balloon in the artery, pressure may begin to form on the surrounding brain tissue and nerves.[6] Another side effect that is not common in smaller aneurysms, but is typical in large aneurysms, is double vision due to the aneurysm pressed against the optic nerve.[6]

Most individuals will never know they have an aneurysm if they do not experience symptoms. When an aneurysm ruptures, 25% of individuals do not survive the first 24 hours[5] 25% of people have complications that lead to death six months after a rupture has occurred.[5] Most individuals will make a full recovery with no complications especially if medical attention was sought out immediately.[5] The chance for a person with a ruptured CA to make it to the hospital before dying is high. Around 10-30% of these patients do not make it to the hospital while approximately 30% experience a good outcome after receiving the appropriate treatment. Once an aneurysm has ruptured, there is a 20-50% chance of rebleeding to occur within the first two weeks if the rupture was never treated.[3] When a rupture occurs, the bleeding generally only lasts seconds, but the pressure caused from the bleeding can depress the blood and oxygen supply to the brain potentially causing loss of consciousness or possible death.[7]

The most common place for a CA to rupture or leak is into the subarachnoid space. The subarachnoid space is found between the arachnoid membrane and the pia mater layers of the brain.[7] This type of ruptured aneurysm is also called a subarachnoid hemorrhage (SAH).[7] SAH can lead to a hemorrhagic (bleeding) stroke in which 46% of patients die after the first hemorrhage and 80% after the second hemorrhage.[1] "The incidence of cerebral aneurysm rupture causing SAH is estimated to be 6-16 per 100,000 population, and this accounts for 30,000 cases per year in the United States."[3] The rupture of a CA is the most common cause for a person to suffer from a SAH.[1]

Types of Aneurysms

An aneurysm can be characterized by its size, shape, formation, etiology, and location in the body. There are two main types of cerebral aneurysms: fusiform and saccular.[2] A saccular, or berry, aneurysm is the most common type of CA with 90% of CAs found being saccular.[2] It is given its name because of its appearance. The sac of the aneurysm has a narrow neck or "stem" which gives the aneurysm the appearance of a berry hanging on a vine.[2] The sac portion of the aneurysm gets filled with oxygenated blood that is being moved through the artery. The most common location saccular aneurysms are formed and located are off a main artery or branch of a main artery near the base of the brain.[5]

A saccular CA can be classified even further by its size. A small aneurysm measuring 5mm or less, medium measuring 6 to 14mm, large measuring 15 to 25mm, and giant measuring greater than 25mm. Most cerebral aneurysms are small, asymptomatic, and found incidentally during brain imaging or an autopsy.[3] Inflammatory changes and hemodynamically induced degenerative vascular changes are two common causes of the formation of a cerebral saccular aneurysm.[3] Some acquired risk factors of saccular aneurysms include: hypertension, older age, excessive alcohol consumption, atherosclerosis (hardening) of the arteries, and cigarette smoking[3] Some inherited risk factors for saccular aneurysms include: polycystic kidney disease, arteriovenous malformation, Ehler-Danlos syndrome, female gender, and a family history of aneurysms.[3]

A fusiform aneurysm is another type of aneurysm that is less commonly found in the brain compared to saccular aneurysms. It is described as "...circumferential dilations of an intracranial artery without an ostium or neck."[8] The formation of a fusiform aneurysm is typically due to dissection of an artery or atherosclerosis of the arteries.[2] The main arteries that fusiform aneurysms develop in are the vertebral arteries, basilar artery, and posterior cerebral artery.[8] It is difficult to treat fusiform aneurysms. Because a fusiform aneurysm has no formed 'neck' like a saccular aneurysm, coil embolization cannot be used for treatment.[8] Endovascular procedures are highly recommended as treatment options. Stent-assisted coiling (SAC) or flow diversion devices are performed frequently on fusiform CAs.[8] Surgical clipping is used only if SAC or flow diversion devices will not be effective.

Two rare aneurysms that are typically not found in the brain are mycotic and dissecting aneurysms. Both of these aneurysms are classified based on their etiology (cause or origin).[3] An aneurysm that is formed from infections that weakens the walls of an artery, causing it to bulge is classified as a mycotic aneurysm.[5] A mycotic aneurysm is formed due to a pre-existing weakening in the arterial wall that is worsened by certain blood infections.[7] Some infections that cause mycotic aneurysms include septic emboli, which is common for those who have infective endocarditis (IE), or similar types of central bacterial embolism.[9] Around 0.7% to 5.4% of intracranial aneurysms are mycotic, making them one of the most rare type of CAs.[9] The best method for diagnosis is to use cerebral angiography because it is difficult to diagnose small mycotic aneurysms with Computed Tomography (CT) and Magnetic Resonance Imaging (MRI).[9]

Dissecting aneurysms occur when there is "...a tear along the length of the artery into the inner layer of the artery wall, causing blood to leak in between the layers of the wall."[2] This can cause ballooning out on one side of the artery and can cause blockage or obstruction of blood flow through the artery as the blood flows in between two layers of the artery instead of flowing through the main artery. This type of aneurysm is normally formed due to a traumatic injury, especially if the trauma was to the patient's head. The form of treatment used is determined by the size, shape, and location of the aneurysms.[2]

An aneurysm can rupture, leak, or remain unruptured. When an aneurysm ruptures, a hemorrhagic stroke, brain damage, or even death may occur. Double vision, nausea, vomiting, sensitivity to light, and loss of consciousness can also occur if a CA has ruptured.[5] The chances of a rupture to occur increases when the aneurysm's size is greater than 10mm in diameter.[2] Chances of a rupture to occur is also dependent on the location of the aneurysm or if there is a history of a previous ruptured aneurysm.[2] The smaller (7mm or less) CAs have a lower chance of rupturing due to their small size.[3] When an aneurysm starts to leak small amounts of blood into the brain, it is called a leaking aneurysm or sentinel bleeding. The leaking may cause "warning headaches" that can occur days to weeks before a rupture occurs. However, very few people have warning or "sentinel" headaches prior to a rupture.[5] If the leaking aneurysm is very small, the individual will more than likely have no symptoms or warnings that he/she has a bleed. Unruptured aneurysms typically go undiagnosed due to there being no signs or symptoms. If the aneurysm begins to grow and/or rupture, only than will the individual know that he/she has an aneurysm.[5]

Signs, Symptoms, and Risk Factors

Even though the exact cause of a CA is unknown, an individual may be diagnosed with a CA because of his/her signs, symptoms, and personal health risk factors.[7] CAs can be either asymptomatic or symptomatic depending on the location in the brain.[1] 90% of people with CAs are asymptomatic, or show no symptoms, and the CAs are normally smaller than 10mm in diameter.[2] Because CAs are typically so small, there is a less of a chance for them to rupture due to the small amount of blood flow.[2]

The most common ruptured aneurysm symptom is described as "the worst headache of my life" by those patients who have survived a ruptured aneurysm.[3] It is very sudden and excruciatingly painful. The headache is caused due to blood rushing into the subarachnoid space of the brain. This is called a subarachnoid hemorrhage or SAH. Around 10% to 43% of people who have SAH will experience a mild to moderate headache approximately two months before a rupture and 30% to 50% claim to have sudden onset headaches six to twenty days before a rupture.[3] Some symptoms of a ruptured CA include high blood pressure, bloodstream infections and eye drooping.[1] The risk factors include congenital aneurysms (present at birth), alcohol consumption, and cigarette smoking.[1]

Complications that can occur after a rupture are re-bleeding, vasospasm, hydrocephalus, and hyponatremia.[7] Re-bleeding can cause further damage to the brain cells. A vasospasm (narrowing of the blood vessels) limits the amount of blood flow reaching the brain causing an ischemic stroke and more cell damage. Hydrocephalus occurs when the ruptured aneurysm causes bleeding in the subarachnoid space causing a block of circulation in the subarachnoid space to the fluid that surrounds the brain and spinal cord.[7] This can cause an excess of cerebrospinal fluid which in return increases the pressure on the brain, damaging its tissues and nerves. A drop in sodium levels (hyponatremia) can cause the brain cells to swell and create permanent damage.[7]

The size and location of an aneurysm is also a crucial factor of if the patient's aneurysm(s) will cause signs and/or symptoms . An aneurysm is more likely to cause symptoms if it is larger in size. When an aneurysm is larger in size, it can begin to push up against nerves causing eye pain, vision blurriness or loss of vision.[3] The location of an aneurysm is important because an aneurysm is more likely to rupture in particular vessels than others. "Aneurysms located on the posterior communicating arteries (a pair of arteries in the back part of the brain) and possibly those on the anterior communicating artery (a single artery in the front of the brain) have a higher risk of rupturing than those at other locations in the brain."[5] If an aneurysm is located at a bifurcation (where the artery begins to separate into smaller arterial branches), there is a greater chance of rupture due to the increase of pressure passing through the bifurcation and the aneurysm.[2]

A person's medical/family history can pose risk factors of developing a CA. An individual who has relatives with CAs or a previous SAH are three to seven times more at risk to have a ruptured cerebral aneurysm in his/her lifetime.[3] A list of inherited risk factors include alpha-glucose deficiency, Alpha 1-antitrypsin deficiency, polycystic kidney disease (PCKD), arteriovenous malformation (AMV), Ehler-Danlos Syndrome, family history of aneurysm, and female gender.[2] Acquired risk factors include cigarette smoking, hypertension (high blood pressure), alcohol consumption, and older age.[2] Smoking is found to cause both the development and rupture of CAs. It can also cause multiple aneurysms to form.[5] High blood pressure causes damage and weakening to arterial walls making it easier for a rupture to occur.[5] Overall, these risk factors help to increase the chance of a cerebral aneurysm, but they do not necessarily always cause one to occur; they just allow a greater chance of an individual to develop a CA.[2]

Grading Charts for Ruptured Aneurysms

The severeness of a ruptured aneurysm can be classified by referring to a system called the Hunt and Hent classification system. The Hunt and Hess classification system was created in 1968 and uses five different grades that range the severity of the symptoms to correspond with the overall mortality of a SAH.[3] It is based on the clinical findings of a ruptured aneurysms and it aims to predict the patient's outcome and mortality.[3] There are five gradings found in this scale. They range from grade 1, being asymptomatic with minimal headaches to Grade 5, being a deep coma or mortality.[10] The measuring of each grade is found below in figure 3. This scale is most commonly used by clinicians and researchers to predict the outcome of a patient undergoing a SAH.[10]

The Fischer scale is also used to classify SAH in Computed Tomography scans.[11] It has four grades ranging from no SAH or intraventricular hemorrhage (IVH) found to a detected symptomatic vasospasm. The grading scale is different as the highest grade is grade three with its grade four defining milder symptoms of a SAH or a vasospasm. The Fischer scale also helps predict the chance of a cerebral vasospasm to occur.[11]

A modified Fisher scale was produced from the original Fischer scale to grade CAs ruptures. It also was modified to grade the thickness of cisternal blood and IVH hemorrhage.[11] The scale starts at a grade 0 and goes up to a grade 4, the highest and most severe grade.[11] This scale is used when performing a non-contrast CT. Figure 4 describes and demonstrates the modified Fisher scale and the original Fisher scale.

Diagnosis

If an individual is experiencing severe headaches or are having symptoms that indicate a possible CA, tests will be ordered to see if there is blood that has leaked between the brain and skull.[5] A Magnetic Resonance Imaging (MRI), Computed Tomography (CT), or Digital Subtraction Angiography (DSA), are recommended for individuals who are at a high risk of having a ruptured cerebral aneurysm. This includes people with a family history of an aneurysms and medical conditions that are associated with cerebral angiograms.

The first steps in diagnosing a CA is ordering a Computed Tomography (CT) scan of the brain. It uses radiation and computer technology to produce two-dimensional and three-dimensional images of the body. These images are cross-sectional slices taken both horizontally and vertically of the body. Bone, fat, muscle, and other tissues can be visualized in a CT scan and provide a more detailed view of human anatomy than an x-ray can provide.[1] A CT scan is fast and often the first test a physician orders to determine if or where blood has leaked into the brain.

In some CT scans, contrast dye is injected intravenously during the exam in order to get a better visualization of the arteries. This type of CT scan is called a CTA (computed tomography angiogram). It provides the radiologist and physician a better understanding of the blood flow, size, location, and shape of the aneurysm.[5] The CTA with contrast is most often ordered for a CT head or brain because, “It is 100% sensitive for SAH if it is done within six hours of symptoms onset, but sensitivity decreases over time to 95% in twelve hours, 92% in twenty four hours, and

50% in one week."[3] CTs and CTAs produce a more detailed image than radiographs and are also able to detect more abnormalities such as the approximate location or type of stroke that occured.[2]

A lumbar puncture (LP) can be performed if the CT scan is negative but there is still suspicion of an SAH. During a LP, a spinal needle is inserted into the lower back or lumbar spine of the patient. The needle is placed in between two disc spaces and inserted into the spinal canal. Fluoroscopy is used while placing the needle to ensure the correct placement. Once the needle is placed into the spinal canal, cerebral spinal fluid, or CSF, is drawn and taken to a laboratory to be tested.[5] The medical laboratory technician will test the CSF by measuring the cell count and xanthochromia of the fluid. If the lab results come back indicating a bleed, a CTA, MRA, or DSA are used to find where the bleeding originated. These tests are 100% sensitive when performed within 12 hours to one week of the bleed.[3]

A magnetic resonance imaging test, or MRI, is another commonly used test to diagnose CAs. It uses magnets, radiofrequency, a magnetic field, and computer technology to produce images of the internal structures of the human body.[1] Two and three dimensional images of the brain can be formed with this technology and can show if there is blood leaking in the brain.[5] It can be used to find the specific location and diagnosis of an aneurysm or SAH.

A magnetic resonance angiogram, or MRA, uses both MRI and intravenous (IV) contrast dye to image the blood vessels in the body.[2] The contrast appears opaque on the images to allow the radiologist to see which vessels are being evaluated.[2] This type of imaging can also detect aneurysms that have a chance to rupture based on the "...inflammatory changes and macrophage deposition of occurring at the aneurysm wall…"[3]

Digital subtraction angiography (DSA) or angiogram uses fluoroscopy to visualize and evaluate the blood vessels. The procedure starts by inserting a catheter (thin, tiny tube) into the femoral artery at the groin level. The catheter is then guided superiorly to the arteries in the brain. Contrast dye is then injected through the catheter to make the arteries and the blood flow through the vessels visible.[2] A cerebral aneurysm's size, shape, location, and possible cause of bleeding can be determined during an angiogram. DSAs can also detect blockage in arteries and find weak spots in the walls.[5]

Treatment Options

Once a ruptured aneurysm is discovered, it is important that it is treated as soon as possible due to the chance of rebleeding.[3] There are a few options for treating an CA: surgical clipping, endovascular coiling and embolization, flow diversion, and observation. Figure 5 depicts how each type of treatment works in a saccular aneurysm. Aneurysms can be treated at any stage; ruptured and leaking aneurysms are considered to be an emergency procedure, while a non-ruptured or leaking aneurysm can be treated if the individual and the physician want to treat it as a non-emergency procedure.[6] It is important for the patient and the physician to discuss the safest plan of care for his or her specific case.[2] Patients' age, medical condition, size, and location of the aneurysm should be taken into consideration when choosing the best treatment option. Some other factors include the patients symptoms, the presence or absence of aneurysm risk factors, and a history of SAH.[3]

Surgical clipping of a cerebral aneurysm is done under general anesthesia. The neurosurgeon performing the procedure will perform a craniotomy, which entails making an opening in the skull. Then the neurosurgeon will proceed through the cisterns and fissures of the brain to find the location of the aneurysm. Once it is located, a small metal surgical clip is placed across the neck of the aneurysm to prevent blood from flowing into the sac of the aneurysm.[3] If the clip is placed correctly and seals off all blood flow from entering the artery, the chances of it reforming or leaking are very low.[5]

After a patient is discharged from the hospital, it is preferred that the patient undergoes a comprehensive evaluation to ensure his/her brain is properly functioning after such an invasive procedure.[3] One benefit of treating an aneurysm by surgical clipping is that the chances of a post surgical bleed is lower than other treatments.[3] The chance of a surgically clipped aneurysm to have rebleeding ranges from 0-0.9%, compared to treatment by endovascular coiling which is 2.6%.[3] Even though surgical clipping can be very beneficial, it can still cause the risk of damage to other blood vessels, the recurrence and rebleeding of the aneurysm and the risk of a stroke.[5] The death rate and chance of a permanent disability is more common in those who choose the surgical route compared to the endovascular coiling route.[3]

Aneurysms that are in a difficult location are preferred to be treated by embolization instead of by surgery.[3] Endovascular coiling and embolization are performed similarly to DSAs. It is important that the patient's blood pressure is around 120/80 mmHg before the procedure occurs to prevent the aneurysm from obstructing during the procedure.[3] Aspirin is usually ordered to be taken three to seven days before the procedure in order to help reduce the risk of clotting.

Clopidogrel (Plavix) is usually taken with the aspirin in cases where the aneurysm is located in a difficult position in the brain.[6] Before coils can be placed, the physician will first have to perform a DSA to determine the location, size, shape, and vessel type of the aneurysm.[6] Endovascular embolization, "...is a minimally invasive technique in which a catheter is advanced from the femoral artery into the cerebral aneurysm, and with the use of a second microcatheter, platinum coils are inserted into the aneurysm."[3] This causes the aneurysm to become clotted off, or embolized, and prevents it from rupturing. This procedure can be performed under either general or local anesthesia.[2] After the procedure, the patient will stay overnight for observation and is generally discharged the following morning.

A newer form of treatment for CAs is called flow diversion.[3] It is very similar to endovascular coiling and embolization, but instead of using coils, a small, flexible mesh stent is used. It is made of a self-expanding mesh divide that is placed into the aneurysm.[3] This reduces the blood flow going into the aneurysm. It is inserted through a catheter and released only once it reaches the aneurysm. This type of procedure is normally done to treat large aneurysms and those that cannot be treated by surgery or embolization.[5]

A person with an unruptured aneurysm that is under 7mm in size, older than the age of 64, asymptomatic, and has no family history of SAH may consider the route of observation by clinical staff or radiologic monitoring.[3] If an aneurysm has ruptured, there are less common treatments available to help control symptoms. Anticonvulsants (antiseizure drugs) help prevent the patient from having seizures; calcium channel blocking drugs reduce the risk of a vasospasm stroke, and rehabilitation therapy. Rehabilitation therapy helps individuals who have previously had a SAH. Physical, speech, and occupational therapy can help the individual gain any lost function and or help him/her learn to cope with any possible permanent disability.[5]

Endovascular Embolization Treatment Case Study

A study performed at Paul L. Foster School of Medicine (PLFSM), Texas Tech Health Science Center (TTUHSC) in El Paso, Texas, evaluated the treatment of ruptured and unruptured cerebral aneurysms with endovascular embolization. All of the cases were performed by the neurointerventional team at PLFSM. The grading scales used to assess the severity of a patient's SAH due to a ruptured aneurysm were the Hung and Hess grading scale and the Fisher grade scale. Each participant was treated under general anesthesia. The femoral artery was used for the main method of access. All of the rupture and unruptured aneurysms were embolized by using detachable coils. Balloon remodeling coil embolization was used for some ruptured aneurysm cases, while the stent-assisted coil embolization technique was used for unruptured CAs.

A total of 45 CAs were treated between March of 2011 and March of 2012. 27 of the 45 aneurysms were ruptured, which accounted for 60% of the sample size.[12] The age range was 35-92 years-old with 57 years-old being the mean age.[12] The Hispanic/Latino ethnic group made up 80% of the CAs treated. The study also consisted of patients with comorbidities (more than one disease) that include arterial hypertension (44%), diabetes mellitus (15%), dyslipidemia (9%), and chronic obstructive pulmonary disease (6%).[12]

The majority of the treated CAs were located in the anterior circulation of the brain: the middle cerebral artery (26%), the internal carotid artery (22%), and the posterior communicating artery (20%).[12] The mean diameter of the aneurysms was 8.5mm and ranged from 2.5-28mm. The mean for a ruptured aneurysm diameter was 7.1mm and the mean for an unruptured aneurysm diameter was 9.8mm.[12] The severity of a SAH for a ruptured aneurysm was graded using the three scales previously listed. The results were, “45% of the ruptured aneurysms that underwent endovascular treatment had a Hunt and Hess grading score equal or less than III and almost 70% of the patients had a Fischer scale equal or less than 3.”[12] Figure 6 contains more information pertaining to the population and characteristics of the aneurysms.

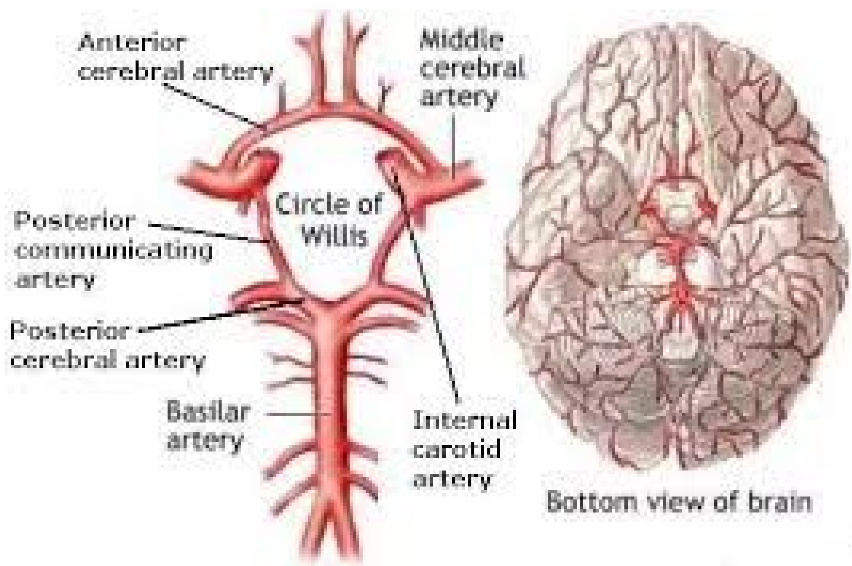
Of the 45 patients treated, seven of the patients (or 15%) had either procedural or periprocedural complications occur, with four being minor complications. The four minor complications were endovascular technical complications that led to no neurological or other clinical detriments. The three major complications included, “a cerebral infarct due to an intraprocedural intracranial dissection during embolization of an anterior communicating artery aneurysm, a posterior cerebral artery infarct in a ruptured posterior communicating artery aneurysm and an intraprocedural aneurysmal rupture during embolization of a ruptured aneurysm resulting in hemorrhagic stroke and death.”[12] The first of the four minor complications was a catheter-induced dissection of the cervical internal carotid artery. The patient had fibromuscular dysplasia and was treated successfully with an acute primary stent placement. The second minor complication was caused by a coil migrating into the distal aspect of the parent vessels. It did not cause a restriction of blood flow or clinical stroke.The third complication was a transient short intraoperative aneurysmal dome perforation that was treated by further coil embolization.[12] The fourth and last minor complication was a fistula in the carotid cavernous and required no treatment due to it resolving spontaneously. More information about each compilation can be seen in Figure 7 below.

The study found that patients who have an aneurysm rupture have an increased rate of morbidity and mortality. It was found that re-rupturing had a high chance of occurring within the first few days to weeks after the first rupture. Because of this, it is recommended to receive treatment shortly after a CA diagnosis. The study also discovered that the risk of having complications intraprocedural is higher in CAs that have ruptured.[12] The team concluded the study saying, "In our condition, endovascular treatment shows good outcomes and some advantages over surgical treatment, at discharge and later survival even compared with other studies." [12]

Conclusion

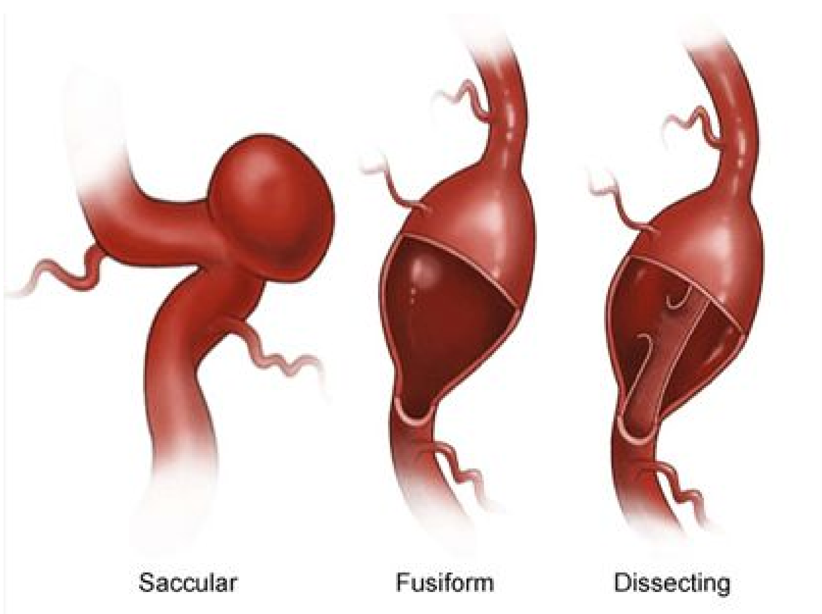
Cerebral aneurysms can go unnoticed for many years or possibly for the individual's entire life before being diagnosed. A saccular aneurysm is the most common type of CA. An aneurysm can be diagnosed if the patient is showing symptoms related to a CA. A person who has a family history of CAs is more likely to have one. CT, MRI, and DSA can all be used to diagnose a brain aneurysm. Based on the diagnosis, a treatment plan is formed by the patient and the provider inorder for the patient to receive the best outcome. The main treatment options available today include surgical clipping, cerebral embolization and coiling, and flow diversion. Each treatment option can help prevent the rupture of a cerebral aneurysm.

Fig. 1 Circle of Willis



Circle of Willis Diagram .; 2017.
https://www.quora.com/In-the-Circle-of-Willis-what-are-the-3-arteries-going-into-6-arteries-leaving-and-3-completing-the-circle. Accessed November 19, 2020.

Fig 2 Depictions of Saccular, Fusiform, and Dissecting Aneurysms



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https://www.rwjbh.org/rwj-university-hospital-new-brunswick/treatment-care/neurosciences/neurosurgery/for-patients/new-jersey-brain-aneurysm-avm-program/what-is-a-brain-aneurysm-/. Accessed December 14, 2020.

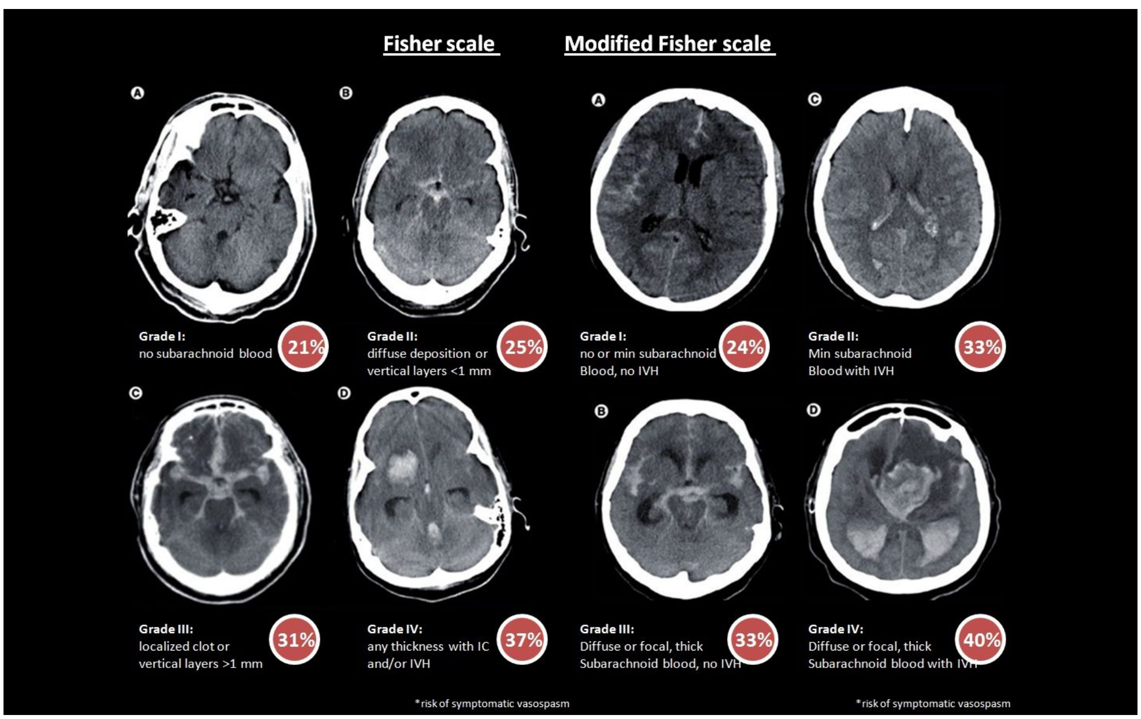
Fig. 3 Chart of Hunt and Hess grading scale for Subarachnoid Hemorrhages

SAH Clinical Grading Scales

Grade	Hunt and Hess	WFNS
0		Intact aneurysm
1	Asxic / mild HA	GCS 15
1a	Fixed neuro deficit s men. or brain rxn	
2	Mod to sev HA, CN palsy, nuchal rigidity	GCS 13–14 no motor deficit
3	Lethargy, confusion, mild focal deficit	GCS 13–14 motor deficit
4	Stupor, hemiparesis, early decerebrate	GCS 7–12 +/- motor deficit
5	Coma, decerebrate, moribund	GCS 3–6 +/- motor deficit

Hunt and Hess Scale for SAH.; 2015. https://pbrainmd.files.wordpress.com/2015/03/sah-grading-scales.jpg. Accessed November 19, 2020.

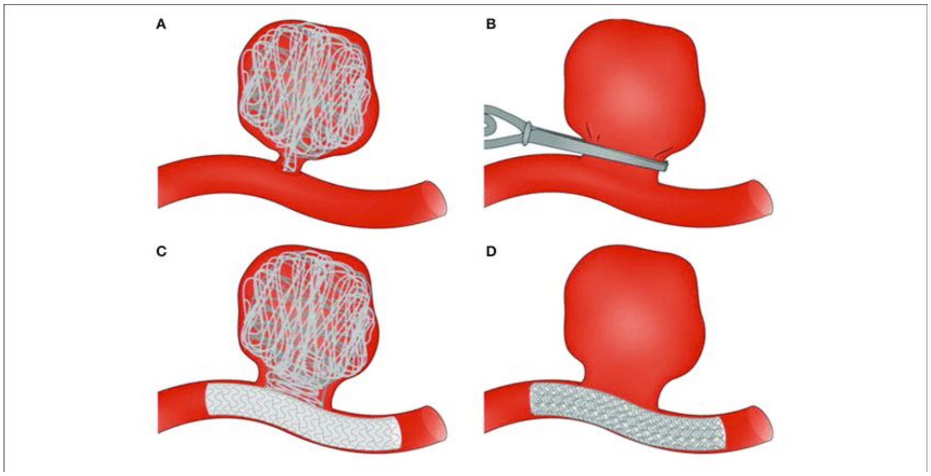
Fig 4 Fisher scale and Modified Fisher scale



Modified Fischer Grading Scale for SAH.; 2015. https://pbrainmd.wordpress.com/2015/03/24/modified-fisher-grading-scale-for-sah/. Accessed December 14, 2020.

Fig. 5 Illustration of different treatment options for saccular aneurysms

Surgical and endovascular treatments for cerebral aneurysm thrombosis. (A) Endovascular coiling of the aneurysm sac. (B) Surgical clipping of the aneurysm neck. (C) Endovascular treatment combining use of coils and a stent. (D) Endovascular treatment with a flow diverter. Taken from Perrone et al. (2015).



Frangi AF, Ngoepe MV, Byrne JV, Ventikos Y. *Surgical and Endovascular Treatments for Cerebral Aneurysm Thrombosis*.; 2018. https://www.researchgate.net/figure/Surgical-and-endovascular-treatments-for-cerebral-aneurysm-thrombosis-A-Endovascular_fig1_324215253. Accessed November 19, 2020.

Figure 6. Table 1: Population and lesion chart

Table 1

Population and lesion characteristics.

	Ruptured (%)	Unruptured (%)
No. cases	27 (60)	18 (40)
Male/female	7/20	5/12
Age range (mean)	35–92 (55, 96)	37–80 (60, 29)
Size of aneurysm		
<7 mm	14 (51, 85)	3 (16, 66)
7–12 mm	12 (44, 44)	13 (72, 22)
13–25 mm	1 (3, 70)	1 (16, 66)
>25 mm	–	1 (16, 66)
Localization		
MCA	11 (40, 74)	1 (5, 55)
ACA	3 (11, 11)	2 (11, 11)
Acomm	3 (11, 11)	3 (16, 66)
Pcomm	5 (18, 51)	5 (27, 77)
ICA	4 (14, 81)	6 (33, 33)
VA	2 (7, 40)	–

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Figure 7. Table 2: Procedural and periprocedural complications

Table 2

Procedural and periprocedural complications

Age	Status	Localization	H&H/Fisher	Size (mm)	Complication
57	Unruptured	ICA	–	7.5	ICA dissection
46	Unruptured	ACA	–	9	Pericallosal artery dissection and ACA cortical infarct
62	Ruptured	Pcomm	1/II	11.5	Posterior cerebral artery infarct
61	Ruptured	MCA	4/III	4	Large intracranial hemorrhage, vasospasm, death
55	Ruptured	ACA	4 /III	7.2	Intra operative rupture
61	Unruptured	Pcomm	–	7.6	Left carotid cavernous fistula
80	Unruptured	Pcomm	–	18	Coil migration

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