Trends and treatment of cerebral aneurysms: Review and case studies

By Tracy Christie, Debra Beveridge, and Diane Pottie

Abstract
Subarachnoid hemorrhage (SAH) often results in devastating neurological deficits requiring hospitalization and loss of independence. This is often a difficult time for patients and their families who are struggling to cope with this sudden illness. Current treatment options include surgical clipping of the aneurysm or endovascular obliteration using Guglielmi Detachable Coils. Our purpose in writing this paper will be to review the factors that determine the choice of treatment, and to discuss how nurses can provide our patients and families with the support and teaching needed during this difficult time.

Review and case studies
A form of Brain Attack, subarachnoid hemorrhage (SAH) happens without warning, often causing death or severely damaging the ability to speak, move, or think. The causative factor is most commonly a cerebral aneurysm, defined as a sac-like dilatation of an arterial wall (Schievink, 1997b). Retrospectively, some patients recall warning signs produced by either a leaking of the aneurysm or by the pressure it exerts on the surrounding structures. Examples may be pain above the eyes, ptosis, a dilated pupil, or localized headache. The classic description, “it was the worst headache of my life,” alerts the health team immediately to the possibility of this diagnosis (Bader, 1997). Despite advances in diagnosis and treatment, the statistics remain unchanged, “12% die before hospitalization; 40% die within one month after the event, and more than one-third of those who survive have major neurological deficits” (Wouter, 1997, p 28). These findings are consistent with the review of SAH by Van Gijn and Rinkel (2001).

In this paper, we will review the current thoughts on the etiology of cerebral aneurysms, pathophysiology, and approaches to treatment. In addition, two case studies will be presented that illustrate the two treatment options available at the Queen Elizabeth II Health Sciences Centre (QEI HSC), Halifax, Nova Scotia. Our aim in writing this paper is to provide the neuroscience nurse with the knowledge needed to support the patient and his/her family and to answer their concerns about treatment options.

Etiology
Many factors have been postulated to explain the formation of cerebral aneurysms. An anatomical characteristic of cerebral arteries predisposes them to the formation of aneurysms. Cerebral vessels have a thinner tunica media, or middle coat, than extracranial vessels, and also lack an external elastic layer. Therefore, as the vessel bulges, the compromised vessel wall becomes even thinner, which would explain the fact that most aneurysms rupture at the point of the dome (Hickey, 2002; Barker, 2002). People with connective tissue disorders such as Ehlers-Danlos syndrome type IV, Marfan’s syndrome, neurofibromatosis Type 1, and polycystic kidney disease, have a predisposition to develop aneurysms (Schievink, 1997a). Bacterial and fungal infections may also cause vessel wall weakness, as the bacteria invade and destroy the vessel wall. There is considerable evidence to suggest a familial tendency as well. Research has suggested that the immediate relatives of patients with SAH have four times the risk of a ruptured intracranial aneurysm than the general population (Schievink, 1997a) however, the need for sibling screening remains a controversial issue (Alberts, Quinones, Graffagnino, Friedman, & Roses, 1995; Schievink, 1997a).

Multiple other factors have also been identified in the development of a cerebral aneurysm. These include:
1. Hypertension
2. Gender (females have a higher incidence than men with a 3:2 ratio)
3. Age (most prevalent between age 35 to 60 years old)
4. Cigarette smoking (this is the most important risk factor and increases with the amount smoked and increases the risk of recurrence)
5. Race (2:1 African Americans: Caucasians)
6. Pregnancy and use of oral contraceptives (20 to 30% of maternal deaths are attributed to aneurysm rupture) (Bendok, Getch, Malisch, & Batjer, 1998; Hickey, 2002; Mhurchu, Anderson, Jamrozik, Hankey, Dunbabin, 2001; Peerless, 1997; Wardlaw & White, 2000.)

Pathophysiology
Cerebral aneurysms usually occur at the bifurcation of the major arteries of the Circle of Willis. The majority of aneurysms develop in the anterior circulation, at the junctions of the internal carotid, posterior and anterior communicating or the middle cerebral arteries. Posterior circulation aneurysms are commonly found on the branches of the basilar and posterior inferior cerebellar arteries, and usually account for 15% of aneurysm location (Hickey, 2002). Aneurysms are classified according to shape and size. Both parameters will be discussed later, as their geometry plays a major role in the treatment options. However, the most significant predictor of outcome is the patient’s condition on arrival at the hospital (Bendok, et al., 1998; Chang & Steinberg, 1998).

Cerebral aneurysms produce symptoms resulting from two scenarios:
1. A large aneurysm acts as a space-occupying lesion, causing symptoms dependent on the location. For example, nerve palsy of cranial nerve III causes ptosis (Bader, 1997).
2. A ruptured aneurysm creates a sudden increase in intracranial pressure (ICP) as blood leaks into the subarachnoid space, spreading into the Sylvian fissures and the basal cisterns. A sudden decrease in level of consciousness (LOC) ensues and, depending on the severity, the Cushing’s Triad (hypertension, bradycardia, and irregular respirations) may be present. The presence of blood may cause an occlusion to normal flow of cerebral spinal fluid, leading to the development of hydrocephalus. As well, the blood acts as an irritant, resulting in an inflammatory response, thereby enhancing cerebral edema and irritating the meninges (Chang & Steinberg, 1998).

This sequence of events is responsible for the clinical presentation: severe acute headache, decreased LOC, nausea and vomiting, neck stiffness, photophobia, hypertension, bradycardia, and seizures. In addition, the patient may present with localizing signs related to the vascular territory affected, such as hemiparesis and aphasia (Mayberg, 1990; van Gijn & Rinkel, 2001). Diagnosis of subarachnoid hemorrhage is made on the clinical presentation and the noncontrast CT scan to demonstrate the presence of blood in the subarachnoid space. A lumbar puncture is done only if the CT scan is negative and in the absence of increased ICP to exclude a warning leak (Markus, 1991). Cerebral angiography may be used to detect anatomical features (Mayberg et al., 1994). At the time of presentation, the clinician will assign a “grade” according to a classification system. The Hunt-Hess classification system (See Figure One), the Fisher Kissler grading scale, the World Federation of Neurological Surgeons Scale (WFNSS), and sometimes the Glasgow Coma Scale are used (Oshiro, Walter, Piantadosi, Witham, & Tamargo, 1997; van Gijn, Bromberg, Lindsay, Hasan, & Vermeulen, 1994; van Gijn & Rinkel). The grading system is used to diagnose the severity of the SAH and as a baseline when monitoring for changes in the patient’s condition.

**Treatment options**

Patients who survive the initial rupture of a cerebral aneurysm, and often their family members as well, are presented with the decision-making dilemma of “what to do” following the bleed. One cannot emphasize enough the importance of the neuroscience nurse in being knowledgeable about the treatment options available for patients diagnosed with either a symptomatic or asymptomatic cerebral aneurysm. Patients and their families look to the neuroscience nurse for knowledge and support during this overwhelming and difficult time.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Descriptive criteria</th>
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<tr>
<td>I</td>
<td>Alert, oriented, and mild headache</td>
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<tr>
<td>II</td>
<td>Alert, oriented, moderate to severe headache, stiff neck, cranial nerve palsy</td>
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<tr>
<td>III</td>
<td>Lethargic, confused, minor focal deficits</td>
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<tr>
<td>IV</td>
<td>Stupor, moderate to severe focal deficits such as hemiplegia</td>
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<tr>
<td>V</td>
<td>Comatose, severe neurological deficits e.g. abnormal flexion/extension (Hickey, 2002).</td>
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When a patient is diagnosed with a symptomatic cerebral aneurysm, the primary goal of treatment is preventing further rupture. At the QEII HSC treatment options for both symptomatic and asymptomatic cerebral aneurysms include surgical clipping or endovascular obliteration utilizing Guglielmi Detachable Coils (GDC) which may require stenting or balloon techniques to assist the coiling procedure.

Over the last several decades, surgical clipping has been the mainstay of treatment of both ruptured and unruptured cerebral aneurysms (deGans, Nieuwkamp, Rinkel, & Algra, 2002; Whitfield, & Kirkpatrick, 2001). This treatment involves repairing the cerebral aneurysm via a craniotomy. During surgery, the aneurysm is clipped or a clamp is placed at the base, or neck of the aneurysm, which prevents blood from entering the area (Bendok et al., 1998).

The introduction of GDCs has revolutionized the endovascular treatment of intracranial aneurysms and is rapidly gaining popularity as an alternative approach to surgical clipping in selective cases (Debrun, Aletich, Kehrli, Misra, Ausman, & Charbel, 1998; Flemming, Brown, & Wiebers, 1999; Guglielmi et al., 1992). Since 1997, the division of neurosurgery at the QEII HSC has been offering this treatment option. GDC obliteration involves a general anesthetic, after which a radiologist inserts a guidewire through the femoral artery, and then injects radio-opaque dye to aid in visualization. The guidewire sits outside the neck of the aneurysm until the radiologist is ready to detach the coils. The coils are added into the aneurysm sac until it is filled (Guglielmi et al., 1992). Patients and family members need to be aware that coiling is an invasive procedure with associated risks. The greatest risks during the coiling procedure is the development of microemboli and rupture (Bader, 1997). As well, repeated follow-up evaluations with angiography are necessary. In contrast, aneurysms that are surgically clipped only require an initial follow-up.
The criteria for selecting patients to be treated using GDCs are continually being refined. Currently, the geometry and location of the aneurysm, and the clinical status of the patient affect the indications for, and likely success of, endovascular obliteration (Flemming et al., 1999). Endovascular therapy is more favourable in smaller aneurysms (diameter, four to 10 mm), with neck widths less than 5 mm and dome-to-neck ratios greater than two, in which the chances of total angiographic occlusion are highest (Debrun et al., 1998). In addition, most cerebral aneurysms in the posterior circulation and a few in the anterior circulation are better treated using the endovascular coiling technique rather than surgical clipping. (Dovey, Misra, Thornton, Charbel, Debrun, & Ausman, 2001). One example would be the basilar tip aneurysm. This type of aneurysm is deeply located, confined in a small surgical space, and is surrounded by many peripherating vessels. Endovascular obliteration using GDCs has been accepted as a valuable therapeutic alternative to surgical clipping of basilar tip aneurysms.

Patients diagnosed with an aneurysm located in the anterior circulation are best treated surgically. Anterior circulation aneurysms, including middle cerebral artery (MCA) trifurcation aneurysms, and the posterior communicating artery aneurysm (PcomA), generally are easy to access surgically. The branching vessels can obscure visualization of the aneurysm neck, increasing the risk of coil protrusion into the parent artery or an adjacent branch vessel. Other factors, such as advanced age and associated poor medical conditions, are also considered when selecting patients for coiling using GDCs, but aneurysm geometry and location remain the primary criteria for selection (Holness et al., 2001).

The following two case presentations illustrate the two different treatment options. Both patients were assessed with Grade 1 SAH with no vasospasm. One patient underwent surgical clipping, and the other endovascular obliteration utilizing GDCs.

Case study one

In the spring of 2001, Mr. W.fainted in the bathroom of his home. Upon falling, he hit his head on the bathtub. He had a headache and vomited, which was thought to be secondary to the fall. However, the next day his headache and vomiting persisted and he went to his local hospital for assessment. A CT scan showed a Grade 1 SAH with blood in the basal cisterns and no hydrocephalus. He had a history of high blood pressure and was transferred and admitted to the QEII HSC. The same day he had an angiogram and coiling of the right anterior communicating artery (AcomA). Nurses monitored for changes in neurological status, for signs of microemboli, the groin site for bleeding, distal pulses, and vital signs. They administered intravenous fluids and medications as ordered to keep the patient normotensive. Mr. W. had an uneventful recovery. There was no sign of hydrocephalus and no re-bleed. He was discharged two weeks later with follow-up appointments scheduled for three months, six months and one year post-treatment. Following discharge he felt well, but had a persistent labile blood pressure, e.g., 186/96, which required ongoing monitoring and teaching regarding medications and lifestyle changes.

At his six-month post-operative follow-up appointment, Mr. W.'s angiogram showed compaction of the coil mass in the dome of the aneurysm with growth of the neck, but no evidence of extrusion of the coil mass from the aneurysm. Persistent high blood pressure was felt to have contributed to the compaction. He was later re-admitted and the neck of the aneurysm was recoiled successfully and he was discharged two days following the procedure. Both Mr. W. and his family required considerable psychological support from the nurses, as they were concerned about the need for this follow-up procedure. However, they were pleased with the eventual outcome and were willing to continue to work on controlling Mr. W.'s blood pressure.

Case study two

Mr. A. had a sudden severe headache with mild neck stiffness and transient photophobia. Four days later he was examined. A CT scan showed a lobulated MCA aneurysm on the left with no obvious blood in the subarachnoid space. Mr. A. was a nonsmoker, alert and orientated, with normal cranial nerves, and a BP of 140/80. The following day he underwent a surgical clipping of the left MCA aneurysm. There was an intraoperative rupture of the aneurysm that lasted four minutes and resulted in low BP. An angiogram performed post-operatively showed good clipping with no vasospasm. Nursing care priorities pre-operatively included reducing external stimuli to prevent re-bleeding. Post-operative care included ongoing monitoring of his neurological status including signs of increased intracranial pressure that would indicate re-bleeding, assessment of vital signs and electrolytes, administration of parenteral fluids and medications, and psychological support of the patient and his family. Mr. A. had an uneventful post-operative recovery and was discharged home a week later. At his one month follow-up, Mr. A. was doing well and had no reading or speech impairments.

These case studies demonstrate the treatment options for cerebral aneurysms, either following SAH or based on incidental findings. A recent study at the QEII HSC compared the two treatment options in terms of cost analysis. Results of the three-year retrospective chart review of patients diagnosed with an asymptomatic aneurysm who underwent surgical clipping or GDC obliteration, showed that patients treated with GDC obliteration had a significantly shorter length of hospital stay than patients treated with conventional craniotomy. However, the potential cost savings were offset by the increased cost of the GDC procedure itself, so that on a treatment basis, there was no difference in total cost between the two approaches (Christie, 2002).

In a research study conducted by Holness and colleagues (2001), treatment options were evaluated. This study has been used as a guide to set the standards of cerebral aneurysm treatment options at QEII HSC. The success and safety of coiling for basilar tip, superior cerebellar, ophthalmic/cavernous, and internal carotid aneurysms make it the preferred method when there is a favourable neck/dome ratio. MCA aneurysms usually have morphology that dictate a direct approach and are best treated surgically. Fifty per cent of ACA aneurysms had morphology that did not favour coiling. PcomA aneurysms appeared to have the highest incidence of residual necks. In young patients, surgery may be a better alternative. The success of any approach to the treatment of intracranial aneurysms must be gauged by clinical and radiological outcomes.

Conclusion

The sudden onset of symptoms and possible serious side effects make individuals with SAH emotionally vulnerable. Education of patients and family members about SAH is important, and the causes and treatment options need to be well-explained by the interdisciplinary health team. The patient and the family members are often overwhelmed by the
The neuroscience nurse has a responsibility to become familiar with the treatment choice as it relates to each individual patient. Care for individuals with SAH requires astute ongoing assessment. The nature of the assessment will vary, depending on the severity of the bleed and whether it was discovered as an incidental finding. Assessment needs include motor, sensory, cranial nerve, and cognitive functioning. Early symptoms of vasospasm may be subtle, and information from family and friends may provide the first clues about alterations in mental status. Neuroscience nurses play a vital role in providing ongoing reinforcement through both conversations and written documentation. Care for individuals with SAH requires a multidisciplinary approach, with the treatment choice as it relates to each individual and overall outcome in patients with aneurysmal subarachnoid hemorrhage.

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Tracy Christie, RN, graduated from the General Hospital School of Nursing of Newfoundland in 1997. Tracy has been an active CANN member and enjoys working as a Research Coordinator for Spinal Cord and Cerebral Vascular Disease research projects. Debra Beveridge, RN, CNCC, graduated in 1974 and is a staff nurse in the neurosurgical IMCU and ward at the QEII Health Science Centre, Halifax, Nova Scotia. Diane Pottie, RN, graduated in 1969, became a neurosurgical nurse, and is a staff nurse on the same unit. She is also the founder of SCIP (Spinal cord injury prevention) N.S.

References


