Aneurysmal Subarachnoid Hemorrhage

It is estimated that subarachnoid hemorrhage (SAH) from ruptured cerebral aneurysm affects approximately 30,000 Americans each year. More than one-third die within the first month despite optimal treatment, and, of those who survive, approximately half will have significant neurologic deficit (Mayberg et al., 1994). SAH occurs when a vessel on the surface of the brain ruptures, forcing blood into the subarachnoid space (American Stroke Association, 2004).

SAH affects younger people more frequently than other types of strokes (Johnston, Selvin, & Gress, 1998). The estimated lifetime cost per person for SAH is $228,000, which is higher than other causes of stroke (Taylor, Davis, Torner, Holmes, Meyer, & Jacobson, 1996). Because of the significant morbidity and costs associated with aneurysmal SAH, nurses need to be knowledgeable about treatment, complications, and ongoing care of patients and their families.

MANAGEMENT OF SUBARACHNOID HEMORRHAGE

Aneurysmal SAH is a medical emergency. Prompt recognition and treatment is paramount to optimize the patient’s recovery. The usual presentation is rapid onset, severe headache that may or may not be accompanied by a brief loss of consciousness, nausea, vomiting, and neurologic deficits. A computerized tomography scan is the standard for diagnosing SAH (Mayberg et al., 1994). After a diagnosis of SAH has been made, a cerebral angiogram is performed to determine the source of the bleed (King & Martin, 1994).

The initial hemorrhage is the most significant cause of poor outcome after aneurysmal SAH. However, rebleeding and cerebral vasospasm are complications that threaten full recovery for patients who survive the initial hemorrhage. Surgical clipping of the aneurysm is the recommended course of treatment to prevent rebleeding (Mayberg et al., 1994). Recent studies show promising results for using coil embolization to secure the aneurysm, especially when surgery is “impossible” or “high risk” (Johnston et al., 2002; Brilstra, Rinkel, van der Graaf, van Rooij, & Algra, 1999).

Cerebral vasospasm continues to be a major threat to recovery during the first 2 weeks after the bleed (Ullman & Bederson, 1996). Cerebral vasospasm is defined as sustained narrowing of cerebral blood vessels resulting in decreased flow to the brain that may result in ischemia and infarction (Barker, 2002).

The Writing Group of the Stroke Council of the American Heart Association (Mayberg et al., 1994) put forth guidelines for the management of aneurysmal SAH that continue to be the basis for treatment of patients with SAH. It is recommended that vasospasm be treated with hypertension, hypervolemia, and hemodilution (H/H/H) to prevent and treat cerebral ischemia. Cerebral blood flow is decreased after SAH and the brain’s need for perfusion is dependent on blood pressure. The goal of H/H/H therapy is to provide pressure and volume to promote cerebral blood flow, thereby improving perfusion (Ullman & Bederson, 1996). H/H/H therapy increases the patient’s risk for adverse events such as “cardiac failure, electrolyte abnormalities, bleeding problems, and rupture of an unsecured aneurysm” (Mayberg et al., 1996, p. 12).

Hyperdynamic therapy is an alternative to H/H/H for the treatment of vasospasm. This therapy aims to improve brain perfusion by increasing cardiac output rather than blood pressure and volume (Warnell, 1996). During this phase, the patient should be closely monitored in the intensive care unit. Thorous serial neurologic assessment can alert the nurse and physician to pending or worsening ischemia due to cerebral vasospasm. The use of nimodipine, a calcium channel blocker, is also recommended from onset through day 21. Nimodipine has been shown to reduce poor outcomes after SAH, although the exact mechanism is unknown (Bayer Corporation, Pharmaceutical Division, 1995).

Other complications of SAH are hydrocephalus, hyponatremia, and seizures (Mayberg et al., 1994). Hydrocephalus occurs as a result of blood in the subarachnoid space that is not reabsorbed.
which then obstructs the flow of cerebrospinal fluid (Barker, 2002). Hyponatremia is frequently seen in patients with aneurysmal SAH and may result in hypovolemia, which may further contribute to cerebral ischemia (Mayberg et al., 1994). Prophylactic use of anticonvulsants is recommended in the acute phase after SAH to prevent the risk of seizures that can contribute to rebleeding (Mayberg et al., 1994).

Nurses caring for patients and their families after SAH must be attentive to subtle changes in neurologic status. Neurologic signs related to vasospasm often vary from assessment to assessment (Barker, 2002). This can be frustrating for the patient and family, who are often relieved that the patient has survived the initial event but are not prepared for the unrelenting changes that are common after SAH. The nurse needs to provide ongoing, consistent, and repetitive information to the patient and family about expected complications after SAH to better prepare them for the onset of cerebral vasospasm and the neurologic changes that accompany it.

Patients who survive the critical phase of SAH after aneurysmal rupture will have a lengthy recovery. The patient requires diligent attention to neurologic status related to vasospasm, which can occur up to 21 days after the initial bleed. The primary goal after the critical phase is to prepare the patient to return to his or her highest level of function and be discharged home (Barker, 2002). Patients may experience persistent headaches, fatigue, insomnia, and depression that can be disabling (Buchanan, Elias, & Goplen, 2000). During this time, families need assistance and support to come to terms with the financial, physical, and emotional burden SAH poses.

PREVENTION

Although aneurysmal SAH has been studied extensively, morbidity and mortality for affected individuals remains high. Recognition and modification of risk factors is critical. Risk factors reported to impact SAH are cigarette smoking, hypertension, alcohol use, estrogen deficiency, and family history of SAH. Cigarette smoking is shown to have a strong link to SAH (Kissela et al., 2002). Providing patient education to assist in the control of risk factors could reduce the incidence of SAH (Mayberg et al., 1994; Kissela et al., 2002). The nurse has a pivotal role in providing ongoing education and support for patients and families to modify their risk factors.

REFERENCES


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