

Subarachnoid Hemorrhage in Children: A Nursing Perspective

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ABSTRACT: Spontaneous subarachnoid hemorrhage occurs less frequently in children than in adults. Overall incidence is estimated at less than 5%. Lesions implicated in children are similar to those in adults but have unique aspects. Lesions seen include arteriovenous malformations and aneurysms, although other causes exist. Examination of four different causes of spontaneous subarachnoid hemorrhage in children through case studies highlights similarities and differences in presentation, diagnosis, treatment, nursing problems, and outcome.

Subarachnoid hemorrhage (SAH), a catastrophic event, occurs in all age groups but presents a unique challenge in the age group 18 years of age or younger. SAH in this population is rare, with less than 5% of all SAH occurring before adulthood.

A subarachnoid hemorrhage is the accumulation of blood (usually arterial) below the arachnoid on the surface of the brain. The hemorrhage typically occurs as a sudden burst rather than a slow accumulation. As blood moves into the subarachnoid space, it mixes with cerebrospinal fluid and circulates over the convexity of the brain and spinal cord, which accounts for many of the symptoms associated with SAH.

Classification of SAH is based on the origin of the hemorrhage. SAH may be traumatic (hemorrhage occurs when arteries on the surface of the brain are stretched or lacerated during a traumatic event) or spontaneous (occurring without traumatic origin, usually from an abnormality in the brain parenchyma or from an abnormal vessel). Traumatic SAH is more common in children. SAH may originate from an arterial bleed on the surface of the brain (primary SAH) or from a deeper bleed; blood dissects through brain parenchyma into the ventricular system or subarachnoid space (secondary SAH). Spontaneous hemorrhage tends to be secondary, while traumatic hemorrhage tends to be primary. For purposes of this paper, only primary or secondary spontaneous SAH, occurring in the population 18 years of age or under, will be discussed.

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Causes of SAH

Arteriovenous malformations (AVMs) are by far the most common cause of SAH in children, accounting for about 51% of SAH in this age group. Aneurysms in children are much less common than AVMs, but are much more prone to rupture, accounting for 25% of all SAH in children. Hypertensive cerebral vascular disease, brain tumors, or other abnormalities in the brain account for 9% of SAH in children. Systemic disease, particularly hematologic disorders such as hemophilia, sickle cell disease, or leukemia, account for 5% of SAH in children. Interestingly, 10% of SAH in children cannot be traced to an identifiable cause.

Comparison of causes of SAH in children vs adults reveals that in adults, aneurysms predominate (Table 1). AVMs account for only 6% of all SAH in the adult population. Hypertensive cerebrovascular disease is more common in adulthood, accounting in part for the higher percentage of other neurologic disease causing SAH in adults. Lastly, SAH in adulthood is less likely to be associated with a specific cause than in childhood.

Arteriovenous Malformation

The primary cause of SAH in children is AVM, with slightly more than half (51%) of all SAH attributable to this cause. There is thought to be a greater tendency in the childhood population toward hemorrhage; in contrast, adults most commonly present with seizures from AVM rather than hemorrhage. Although AVMs are often anatomically smaller in children, they are more prone to early second hemorrhage, leading to a greater mortality rate from AVM in children than in adults. AVMs are also more frequently subtentorial in children; 10% to 20% of AVMs in children are subtentorial, compared with less than 5% in adults. The older child is more prone

to develop symptoms from AVM; 85% of AVMs in children are diagnosed in the 7 to 13 years of age group.²

AVMs are thought to develop primarily on a congenital basis and are sometimes associated with other congenital anomalies. AVMs develop embryologically when the normal cerebral vasculature is developing. The capillary system fails to differentiate, and there is direct shunting of blood from arteries to veins, bypassing the capillary network.¹⁸ (The AVM is an embryonic vasculature that lacks a capillary network.) Without a normal capillary network, the function of tissue perfusion is bypassed. Inadequate tissue oxygenation results in cerebral ischemia. Because blood is shunted directly from arteries to veins, a state of altered cerebral vascular resistance occurs.¹⁵ The AVM offers the blood a pathway of less resistance than does a normal capillary network. Hence, arteries dilate to bring more blood to the lesion, and veins dilate to carry the excess blood away. Collateral vessels develop and add to the evolving mass. Cerebral blood flow (CBF) through the lesion may be increased 50% to 100% above normal. Adjacent brain tissue is robbed of its normal flow, a phenomenon known as "intracerebral steal." Blood is diverted from the normal area of the brain to the vascular abnormality, an area of lower vascular resistance.²⁰ AVMs may become symptomatic as a result of this phenomenon or from SAH. The AVM may hemorrhage because its vessels are thin walled and under a great deal of pressure.

If the AVM becomes large, local and systemic involvement can occur. Local involvement is signaled not only by intracerebral steal but also by the presence of the AVM acting as a space-occupying lesion. Systemic involvement occurs with dilation of the arterial trunk of the AVM moving toward the heart. As the circulatory demand of the AVM increases, cardiac output must increase, potentially precipitating heart failure.¹⁸

Aneurysms in Children

Aneurysm as a cause of SAH in children is rare; aneurysms in childhood account for 1.3% of aneurysms for all ages. Specific incidence of aneurysm by age is 0 to 10 years, 15%; 11 to 14 years, 30%; 15 to 20 years, 55%. Aneurysms in children are predominant in males; female incidence predominates for all ages. As in adults, childhood aneurysmal symptoms most commonly are attributed to SAH; rarely do childhood aneurysms present as space-occupying lesion. The anatomic location of childhood aneurysms is unique in that over 20% of cases occur at the carotid bifurcation, whereas in adults, the percentage is about 8% in this location.

Interestingly, a number of cases have been reported in the literature describing children with

Table 1
Causes of SAH-Child vs Adult

	Child	Adult
Arteriovenous malformation	51%	6%
Aneurysm	25%	51%
Other neurologic disease	9%	15%
Systemic disease	5%	7%
No identifiable cause	10%	22%

aneurysms having an associated unrepaired coarctation of the aorta. Children with cyanotic heart disease and subacute bacterial endocarditis (SBE) are prone to bacterial embolization as a cause of aneurysm. Also, morbidity and mortality rates associated with SAH from aneurysm in children are more favorable than in adults in spite of the tendency toward an early second hemorrhage.¹¹

Other Causes of SAH

While AVM and aneurysm are the primary causes of SAH, other causes have been identified. Vascular malformations of the spinal cord may result in SAH. Spinal cord malformations are usually located in the mid- to lower-thoracic and upper-lumbar regions of the cord. They often become symptomatic during childhood, and the onset of hemorrhage may be precipitated by trauma or exercise. Involvement of ascending and descending pathways of the spinal cord leads to motor weakness and sphincter disturbances. There is usually no change in the level of consciousness.⁷

Brain tumor (particularly pituitary adenoma, hemangioblastoma, and intraventricular tumors) and abscess can also cause SAH, but both have a more gradual onset of neurological involvement. Often, prior to the hemorrhage, the patient experiences headache and vomiting in addition to other signs of increased intracranial pressure. Arteriosclerotic vessels have also been identified as a source of SAH.²⁰ Systemic diseases, especially those affecting coagulation, may precipitate SAH in the child or adult. In children, hemophilia, idiopathic thrombocytopenia, purpura, disseminated intravascular coagulation, and sickle cell disease have all been associated with SAH as complications.² Central nervous system leukemia in children may also cause SAH.

Table 2
Assessment of Patients with SAH

Grade 1	Conscious with signs of subarachnoid blood
Grade 2	Drowsy without significant deficit
Grade 3	Drowsy with a neurological deficit
Grade 4	Deteriorating with major neurological deficit
Grade 5	Moribund with extensor rigidity and failing vital signs

From Botterell EH et al: Hypothermia and interruption of circulation in the surgical management of intracranial aneurysms. *J Neurosurg* 1956; 13:1-42.

Table 3
Assessment of Patients with SAH

Grade I	Asymptomatic, or minimal headache and slight nuchal rigidity.
Grade II	Moderate to severe headache, nuchal rigidity. No neurological deficit other than cranial nerve palsy.
Grade III	Drowsiness, confusion, or mild focal deficit.
Grade IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances.
Grade V	Deep coma, decerebrate rigidity, moribund appearance.

From Hunt W, Hess R: Surgical risk as related to time of intervention in the repair of intracranial aneurysms. *J Neurosurg* 1968; 28:14-20.

Symptomatology

Overall symptomatology associated with SAH is consistent among all age groups, with individual variations depending on the site and extent of hemorrhage. Premonitory symptoms, such as localized headache with nausea and vomiting or even visual disturbances, occur from days to weeks prior to the hemorrhage in almost 60% of cases of SAH.¹² Classically, the child with SAH presents with an acute onset of excruciating headache, followed by neck pain and stiffness and/or back pain and stiffness. Vomiting with or without nausea, photophobia, restlessness, and irritability are common. Changes in vital signs can occur, including mild elevations in blood pressure and temperature as well as a decrease in heart rate. Focal signs of neurological dysfunction (ie, weakness, paralysis, cranial nerve palsy, etc) or decreased consciousness may or may not be present. Bleeding into the ventricular system often presents with progressive stupor. Severity of the hemorrhage may be evaluated using the Botterell system (Table 2) or the scale by Hunt and Hess (Table 3).^{3,6} Partial or generalized seizures signaling SAH are not common in children but may occur. In addition, symptomatology may be seen in the absence of SAH as a result of cerebral ischemia (in AVMs, due to intracerebral steal) or the presence of a space-occupying lesion.

Evaluation and Management

The child presenting with symptoms indicative of SAH requires a comprehensive diagnostic evaluation. A CT (computed tomography) scan (with and without contrast) is necessary to document the presence and location of SAH. Source of the hemorrhage (ie, aneurysm or AVM) may be identifiable on the contrast scan. Angiography is essential to more clearly identify the source of hemorrhage and to outline the extent of vascular abnormality present. Vasospasm may also be documented via angiography. Lumbar puncture is not used except where symptoms of SAH exist without identification of the

Table 4
Clinical Guidelines for Hypervolemic Hemodilution

Initial Patient Evaluation

- Establish baseline motor and speech function for subsequent comparisons during therapy.
- Detect altered level of consciousness, which may indicate increased ICP and thereby preclude use of hypervolemic hemodilution.
- Examine cranial CT scan for evidence of acute structural cerebral damage (hypodensity, enhancement, hemorrhage, and/or mass effect), etiology of present insult, and presence of previous cerebral infarctions.
- Examine cerebral angiogram for etiology of present insult and degree of collateralization.
- Determine baseline hematocrit, central venous pressure and, in cases of elderly or cardiac patients, pulmonary wedge pressure or cardiac output.

Infusion Technique

- Intravenously infuse 250 mL of 5% albumin over 30 minutes.
- Adjust infusion rate to maintain reduced hematocrit at about 33 (usually requires 250 mL of 5% albumin every four hours).
- Adjust infusion rate to "normalize" intravascular volume (central venous pressure less than 12 cm H₂O).
- In elderly or cardiac patients, adjust infusion rate to maintain stable cardiac output and pulmonary wedge pressure below 20 mm Hg.
- If level of consciousness deteriorates, stop infusion and perform cranial CT scan. If midline shift has increased, give 25 g intravenous bolus of mannitol. Maintain infusion for three to four days, then taper.
- If improved neurological status worsens during infusion taper, increase rate and reattempt taper in two to three days.

Adapted from Wood J: Hypervolemic hemodilution: Rheologic therapy for acute cerebral ischemia. *J Neurosurg* 1982; 10:781.

hemorrhage on CT scan. Other diagnostic testing may be required, depending on individual situations.

Regional cerebral blood flow measurements (rCBF) made by xenon inhalation technique can play an important role in evaluating the functional status of the cerebral vasculature. In general, rCBF in patients with SAH has been shown to decrease following the hemorrhage; decreases in rCBF contribute to ischemia and potentially infarction. Decreases in rCBF are attributed to increased blood viscosity and elevated hematocrit. Surgical intervention initiated during the time of decreases in rCBF has been associated with a higher morbidity and mortality.

In an attempt to improve rCBF following SAH, the nonsurgical therapy of hypervolemic hemodilution may be used. (This therapy is thought to be effective in all age groups.) The goals of this therapy are to decrease hematocrit and increase intravascular volume, allowing subsequent improvement of rCBF.¹⁹

These goals may be achieved through the use of albumin. Albumin is a colloid infusate that remains within the intravascular compartment after infusion. In contrast, crystalloid infusates such as saline and dextrose distribute throughout the body with only 15% remaining in the intravascular compartment. Albumin promotes hemodilution by expanding

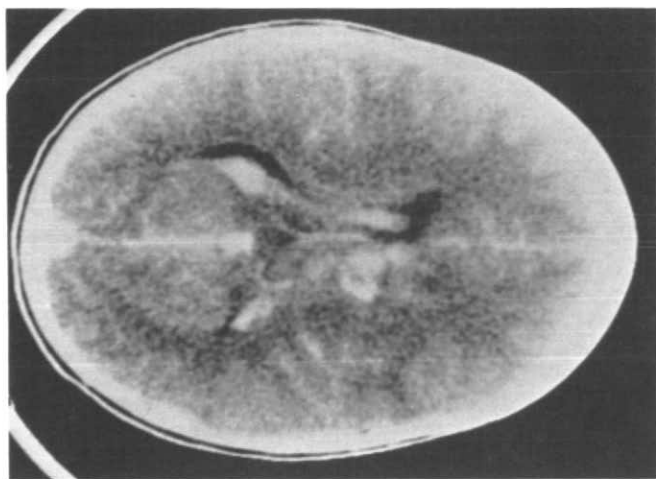


Fig 1. Left thalamic AVM with hemorrhage into left lateral and third ventricle.

plasma volume, thereby decreasing hematocrit.¹⁹ Patients with SAH grades I through IV may benefit from this therapy. Prior to initiating the therapy, baseline hematologic values are reviewed, including hemoglobin (Hgb), hematocrit, erythrocyte sedimentation rate, and albumin and fibrinogen concentrations. Alert patients with a hematocrit greater than 38 and no evidence of increased intracranial pressure or midline shift on CT scan should have the therapy started immediately. Specific guidelines for the therapy as adapted from Wood are listed in Table 4.¹⁹

Problems of fluid overload and pulmonary congestion, which may occur with intravascular volume expansion, are monitored via CVP or Swan-Ganz catheter. Infusion rates are adjusted to maintain the CVP less than 12 cm H₂O and pulmonary capillary wedge pressure less than 20 mm Hg. The hypervolemic hemodilution therapy is usually maintained for three to four days, followed by gradual reduction of the volume of infusions (weaning). Frequent neurological exams are necessary during therapy and tapering of infusions to evaluate any deterioration in the patient's neurological status. Deterioration during weaning may indicate the need to continue therapy. Alteration in the patient's neurological status during weaning may be corrected by increasing the rate of the infusion.

Case Studies

Case #1:

WB, an 11-year-old girl, complained of sudden onset of a severe headache while twirling a baton. This was immediately followed by a decrease in her level of consciousness and three episodes of vomiting. On arrival in the emergency room, WB was stuporous but arousable by vigorous shaking. No seizure activity was noted. She was unable to speak and was found to have a right hemiparesis. Nuchal rigidity was

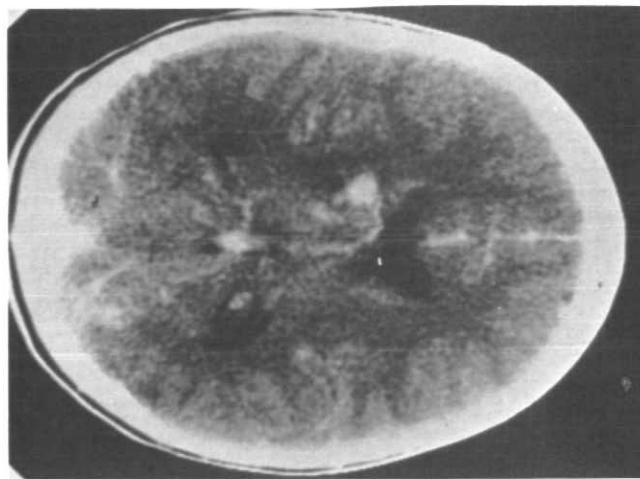


Fig 2. Hemorrhage resolved—left thalamic AVM.

present. Prior medical, social, and academic history was unremarkable, except for a three-month history of mild headaches. According to family, WB was an above-average student and a normal eleven year old. CT scan on admission revealed a left thalamic AVM with hemorrhage into the left lateral and third ventricles (Fig 1, 2). Angiography identified the thalamic perforators and lenticulostriate vessels as feeding vessels; the AVM was drained by thalamic veins (Fig 3). WB's condition improved over the first 24 hours. Only a mild right hemiparesis was present, and speech appeared intact. Although lethargic, WB responded to verbal stimuli. Her alertness steadily improved over the next seven days. Eight days after admission, WB was taken to the operating room for a left temporal-parietal craniotomy and resection of the AVM. The procedure was tolerated well. Post-operatively, WB's right-sided weakness persisted but was now more pronounced in the lower extremities. In addition, a right facial droop with deviation of the tongue to the right was noted. Speech fluency was again a problem. Over the next several weeks, WB

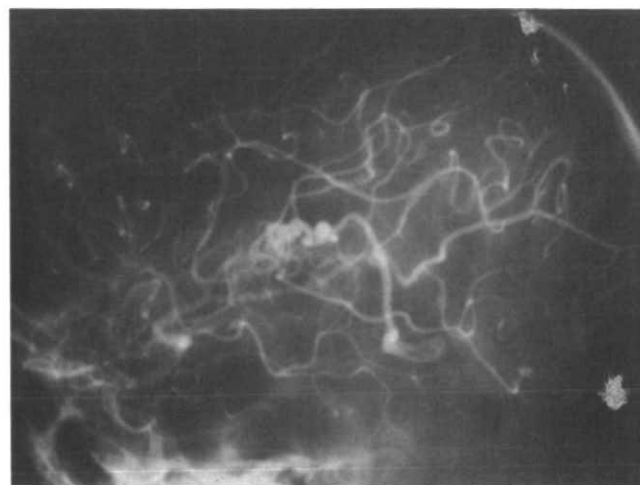


Fig 3. Left thalamic AVM with thalamic perforators and lenticulostriate feeding, draining via thalamic veins.

underwent intense physical therapy rehabilitation. By the time she was discharged from the hospital, nine days after surgery, she was able to ambulate and the right-sided weakness and facial droop had almost fully resolved. WB was to continue outpatient physical and speech therapy on a regular basis and was to be followed up in the outpatient clinic.

Nursing Problems: Although WB's aphasia was transient, it required nursing intervention. A Broca's aphasia was present secondary to the location of her lesion. Her reading and writing skills remained intact. Nursing interventions were directed toward fostering an environment of effective communication. Specific nursing interventions included: 1) listening attentively; 2) anticipating WB's needs; 3) providing support and encouragement for the mobility she had already attained; 4) having only one person at a time communicating with WB and; 5) providing an atmosphere of comfort, calm, and acceptance to help decrease WB's anxieties regarding her communication difficulties. We explained to WB in simple terms why she was experiencing difficulties and this helped her to understand and accept her situation.

The right hemiparesis was present both preoperatively and postoperatively; spasticity was present as well. Prior to developing a plan of care, a thorough assessment of WB's capabilities was necessary. Initially, WB had poor strength throughout, especially in the right arm and right foot. She had fair sitting balance. Standing balance was poor; proprioception was decreased, as was kinesthetic awareness. Specific nursing interventions included the following: 1) an unhurried approach to assist her with mobility, transferring, range-of-motion exercises, and activities of daily living, with adaptations as necessary; and 2) assisting WB to recognize and understand her strengths and weaknesses.

Two months after leaving the hospital, WB returned for a neuropsychological evaluation. By this time she had resumed almost all of her normal activities of daily living. Her speech was fluent, and no language or vocabulary abnormalities were noted. Interestingly, it was noted that WB exhibited a right hemisphere dominance for language, and this was one of the reasons she experienced only a transient aphasia preoperatively and postoperatively. Her neuropsychological evaluation did not suggest any cognitive dysfunction, and she was given a prognosis for normal development. WB has resumed all of her previous activities without limitations.

Case #2:

ST, an 11-year-old boy, presented with complaints of a severe frontal headache that came on suddenly while he was swimming. He went home with the headache and experienced nausea and vomiting for

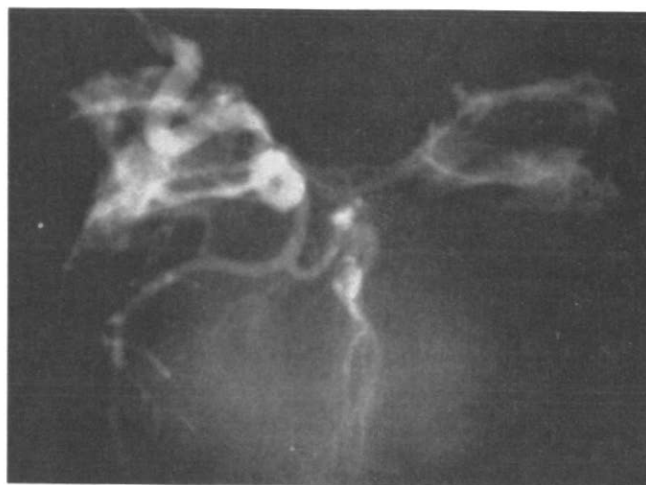


Fig 4. Anterior communicating artery aneurysm.

several hours. Since his symptoms were not relieved with rest or acetaminophen, his mother brought him to the emergency room. On arrival, ST was found to have nuchal rigidity, a positive Kernig's and Brudzinski's sign, and a low-grade fever. Past history was remarkable for generalized moderate headache diagnosed as vascular in origin. A lumbar puncture (LP) was done because of suspicion of meningitis; LP was grossly bloody, and CSF gram stains were negative. CT scan done subsequently documented the SAH. Angiography performed the next day demonstrated an anterior communicating artery aneurysm (Fig 4).

ST was placed in a pediatric intensive care unit for careful observation. Bedrest and sedation were ordered to minimize the chance of a rebleed. ST was kept well hydrated, and the hypervolemic hemodilution therapy was used. Protocols previously mentioned for the therapy were followed. Five days after admission, ST underwent a left temporal-parietal craniotomy for clipping of the aneurysm. He tolerated the procedure well. Postoperatively, ST had no neurological deficit nor any complaints. He was discharged home on the fifth postoperative day. His posthospital period was unremarkable.

Nursing Problems: Altered fluid balance was considered a problem for ST because he was undergoing hypervolemic hemodilution therapy. His fluid balance was monitored with a Swan-Ganz catheter and frequent monitoring of hematologic values. His volume status remained within normal physiologic limits.

The second problem identified was altered psychosocial status secondary to his illness. ST suffered many anxieties and fears related to his hospitalization and diagnosis. Much of his behavior was attention-seeking behavior, in which he demanded much of the nurses' time. He would continually ask a nurse to be at his bedside playing with him, talking to



Fig 5. Third and fourth ventricular hemorrhage.

him, or holding his hand. He especially requested physical comforting. The nursing staff adjusted their schedules to spend time with him and became adept at meeting his needs. The support offered helped him to work through some of his fears and anxieties. One of ST's greatest fears was of hair loss. He became preoccupied with this idea. He was afraid of what he would look like postoperatively and that his hair would never grow back. After surgery it was obvious that ST did not feel good about his body image. ST was encouraged to express his fears; his value as an individual was continually emphasized. He was also encouraged to talk with other children on the unit who had had similar surgery. ST is back in school, doing well, and has resumed all of his previous activities, including swimming. (His hair is fully grown.)

Case #3:

MP, a nine-year-old girl, experienced a severe headache while tumbling in gym class. The headache was followed a few minutes later by a single episode of vomiting and an acute loss of consciousness. She was brought to the emergency room where she was noted to have spontaneous movement but did not follow commands (a Glasgow Coma Score [GCS] of 6). Movement on the right side was less than on the left; a positive Babinski was present on the right. A right facial droop was noted, as well as nuchal rigidity. Her left pupil was at 8 mm with no reaction, while the right was reactive at 2 mm. Past history was remarkable for a two-month history of headaches diagnosed as possible migrainous headache. She was admitted to the pediatric ICU.

A CT scan initially showed third and fourth ventricular hemorrhage (Fig 5). Angiography was essentially normal, revealing no vascular abnormality. Because of the inability to identify a source for the hemorrhage, magnetic resonance imaging (MRI) was done—results were compatible with a thalamic le-

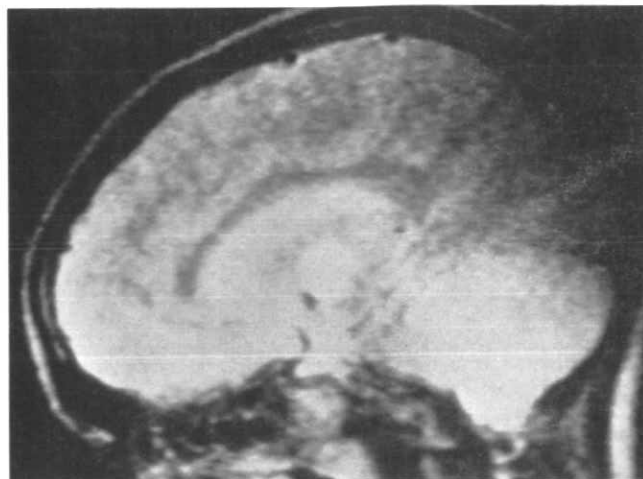


Fig 6. Magnetic resonance imaging of left thalamic lesion.

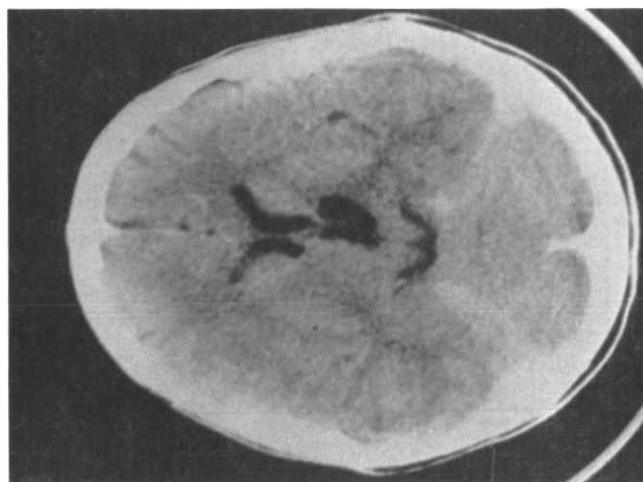


Fig 7. Left thalamic infarct.

sion (Fig 6). A CT scan repeated about nine days later showed a thalamic infarct (Fig 7). Treatment consisted solely of supportive care. Dexamethasone and mannitol were used to treat the mass effect present. Hydrocephalus developed 24 hours after admission, requiring external ventricular drainage for five days. Internalization was not necessary. Because of MP's neurological deficits, physical and occupational therapy were started early.

Nursing Problems: Altered level of consciousness was a problem because MP initially presented comatose. She began to respond consistently on the fourth day after admission, although her level of consciousness fluctuated. Following discontinuation of the external ventricular drainage, there was concern that she would develop recurrent hydrocephalus; the possibility of rebleed also existed. Interventions included: 1) frequent assessment of neurological status with specific observations for trends in consciousness; 2) provision of appropriate sensory stimulation; and 3) implementation of interventions to protect MP

from hazards of immobility.

Altered mobility was a problem once MP's consciousness improved. Specific motor deficits included a spastic right hemiparesis and incoordination. Proprioception was decreased on the right side as well. Interventions included: 1) gradual implementation of range of motion activities from passive to active; 2) gradual ambulation, beginning with transfers from bed to chair, then several steps at a time; and because MP tended to be impulsive, 3) supervision for all motor activities. By discharge, MP was able to ambulate 50 feet with only a near assist.

Family coping deficit was the third problem. MP's mother verbalized feelings of inadequacy as a mother, saying she should have recognized MP's headaches as a more serious problem. MP's mother had recently remarried, and she had thought perhaps MP's headaches were adjustments to her marriage. The nursing staff encouraged MP's mother to become involved in her care. Support services for the mother were identified, and on several occasions she participated in the parents' coffee hour on the unit, discussing her concerns with other mothers.

MP was discharged home three weeks after admission with a mild right hemiparesis. Physical and occupational therapy were continued at home for three months until MP was able to return to school. She now ambulates independently and rides her bicycle, although with rapid activity (ie, running) she is noted to be ataxic. She is doing well in school, although functioning at a slightly lower level. Recent evaluation by a neuropsychologist revealed a mild Wernicke's aphasia, which may be related to her change in grades.

Case #4:

TR, a six-year-old male, woke up in the morning with complaints of mild headache; his mother noted that he was irritable. He sat down to breakfast, vomited, and lapsed into a coma. By the time he arrived in the emergency room, his pupils were at 7 mm and sluggishly reactive. Decerebration was noted to pain. Respiratory irregularities were present, requiring intubation and mechanical ventilation. Past history was remarkable for an 18-month history of acute lymphocytic leukemia, which was thought to be in remission. CT scan showed subarachnoid blood without any specific evidence of intracerebral or intraventricular hemorrhage. Lumbar puncture was done to identify presence of meningeal leukemia. Opening pressure was 270 mm H₂O, and fluid was grossly bloody. Protein was 80 mg%, and sugar was 40 mg%. Cell count was elevated with the differential showing 30 mononuclear cells. (Greater than ten cells is indicative of meningeal leukemia.) Blood studies showed no evidence of active leukemia although thrombocytopenia was present. Replacement

platelets were given, and intracranial irradiation was started (a course of 2000 rads over four weeks). Ten milligrams intrathecal methotrexate was given, to be repeated every two days. Complete supportive care was provided.

Nursing Problems: Altered level of consciousness was a problem—TR's GCS ranged from 3-4. Family and staff were encouraged to provide auditory and tactile stimulation, and his level of consciousness was assessed hourly. Family involvement in decision making related to TR's care was difficult because his parents were separated and often openly disagreed about what action to take. Daily meetings were set up with both parents to give them information; both parents stayed at the hospital 24 hours a day. The hospital risk manager was consulted for legal advice. When the gravity of TR's situation was apparent, attempts were made to prepare his family for the possibility of his death. The dramatic change in his condition was most difficult for TR's family to grasp; blood counts one week earlier showed remission. TR's father refused to discuss any of his feelings, but TR's mother participated in parents' group on the unit. She was referred to the Candlelighters—a support group for parents of children with cancer.

TR showed little response to any of the therapies. On the fifth day after admission, his condition deteriorated to the point where no response was noted. All extremities were flaccid. Pupils were 9 mm with no reaction to light. Signs of cranial nerve function were absent; hypotension and irregular heart rate were present. With the written consent of both parents, a "do not resuscitate" order was instituted. Two hours later, five days after admission, TR died. No autopsy was performed.

Summary

Subarachnoid hemorrhage in children, although rare, presents challenges in diagnosis and management. A comprehensive approach to the unique aspects of SAH in children is necessary. Symptomatology seen in children is consistent with that observed in the adult population. Management of the hemorrhage focuses on an in-depth diagnostic evaluation of the source of hemorrhage, institution of hypervolemic hemodilution therapy to minimize cerebral ischemia, and appropriate surgical intervention where indicated. Nursing problems are unique to the disease and to the individual child. Outcome of SAH in children is variable, from little or no deficit, to substantial deficit or even death.

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