Increased Intracranial Pressure

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Case Study
W.C. is a 50-year-old man initially diagnosed with a glioblastoma involving the right temporal lobe after presenting with headaches and reduced short-term memory in May, 2006. He was placed on corticosteroids and underwent a gross total resection. Following surgery, he received 60 Gy of external beam irradiation with concurrent daily temozolomide at 75 mg/m²; he completed the regimen in August 2006. W.C.’s course was complicated by fatigue, hair loss, and deep venous thrombosis for which he was placed on anticoagulation with Lovenox® (enoxaparin sodium injection, sanofi aventis). He was subsequently placed on adjuvant temozolomide at 200 mg/m² for five days every 28 days.

After completing two months of adjuvant treatment, W.C. presents to the emergency room with a one-day history of increasing headaches and somnolence. This morning, his wife was unable to wake him and called 911. She reports that W.C. fell yesterday while walking his dog, hitting his head on the sidewalk. He did not lose consciousness at the time of the fall. Upon evaluation, he is arousable to sternal rub only and unable to follow commands.

Neurologic emergencies in patients with cancer are frightening and often associated with devastating complications. The episodes can be just as unsettling for the oncology nurse, who may not be familiar with the nervous system or who is witnessing neurologic complications for the first time. Neurologic emergencies can occur in patients with diagnosed primary brain tumors, metastatic lesions, or as a consequence of metabolic or infectious complications in patients with systemic cancer.

Differential Diagnosis
Causes of altered consciousness include increased intracranial pressure, seizure activity, infection, hypoxia, and metabolic abnormalities (Dahlin, 2006). The patient’s clinical history should be obtained from the caregiver. Ascertain the cancer and treatment history, current medications, history of head trauma, and the history of the presenting problem (precipitating factors, onset, pattern, and duration) is important (Dahlin).

Initial evaluation includes assessment of airway, breathing, and circulation; assessment for focal neurologic deficits, and determination of level of consciousness (Vollmer & Dacey, 1991). Brain imaging should be performed as soon as the patient is stabilized (Nolan, 2005).

W.C. had a computed tomography (CT) scan, revealing a large subdural hematoma with midline shift. His physical examination findings and imaging indicate increased intracranial pressure (ICP) (see Figure 1).

Pathophysiology
Common causes of increased ICP are intracranial mass lesions, cerebrospinal fluid (CSF) circulation disorders, and diffuse intracranial pathologic processes (Dunn, 2002). Increased ICP is defined as the pressure exerted by the CSF within the ventricles (Hickey, 1997a). In adults, normal ICP ranges from 5–15 mmHg or 10–20 cm H₂O. Increased ICP is sustained pressure elevations greater than 15–20 mmHg or the presence of intermittent pressure waves (Youmans, 1982). Physiologic elevations of ICP occur with coughing, head-down tilt, Valsalva maneuvers, and compression of neck veins. Although ICP elevations associated with these stimuli are considerable, they do not cause neurologic dysfunction or brain damage because the pressure is distributed equally throughout the length of the craniospinal axis (Miller & Piper, 1985; Thapar, Taylor, Laws, & Rutka, 2001). Other factors that influence ICP under normal circumstances are changes in arterial pressure, venous pressure, intrathoracic pressure, postural changes, blood gases, and body temperature (Muwawas, 1985).

The principal determinants of ICP are the volumes of the brain, CSF, and intracranial blood, with the volume remaining almost constant in a state of dynamic equilibrium (Hickey, 1997a; Hickey, 1997b). Brain tissue makes up the largest component of the intracranial contents and is approximately 1400 ml. The blood and CSF volumes are approximately 75–150 ml each. The intracranial compartment has a special vulnerability because it is a closed system, encased in a rigid
skull. Increased volume of any one of the intracranial components (brain, CSF, or cerebrovascular volume) must be compensated for by reciprocal and equivalent reductions in the other volumes to maintain a steady state. Failure of this volumetric compensatory mechanism leads to increased ICP (Thapar et al., 2001), which can be fatal despite timely intervention (Nolan, 2005).

ICP can be increased by four mechanisms: (a) an increase in intracranial volume (also termed mass effect) as a result of tumor, hemorrhage, or brain edema; (b) increased venous failure as a result of sagittal sinus thrombosis, heart failure, or superior vena cava obstruction; (c) obstruction to the flow or absorption of CSF resulting in hydrocephalus; or (d) pseudotumor cerebri (Victor & Ropper, 2002). Pseudotumor cerebri is an idiopathic condition, most common in young women who are overweight. Patients with this condition are symptomatic with increased ICP, but imaging reveals normal cerebral anatomy.

Increased ICP is a common complication in patients with brain tumors because the mass lesion or associated edema caused by irritation of the cortex around the lesion adds to the volume of tissue in the brain. Focal neurologic symptoms such as hemiparesis, aphasia, or decreased short-term memory can occur and are dependent on the location of the lesion in the brain. Generalized symptoms such as headache and altered consciousness are common symptoms and are a consequence of global neurologic dysfunction (Wen, Schiff, Kesar, Drappatz, Gigas, & Doherty, 2006). Tumors also can cause increased ICP from hydrocephalus, which is the dilation of ventricles caused by a disturbance in CSF circulation (Hickey, 1997a). Brain tumors are the most common cause of hydrocephalus (Miller & Piper, 1985).

Increased ICP from any cause disturbs brain function through disruption of cerebral blood flow or by herniation of the brain. When ICP rises, cerebral blood flow decreases, which results in decreased cerebral perfusion pressure (CPP). CPP is calculated by subtracting the ICP from the mean arterial pressure (MAP) using the formula of CPP = MAP - ICP. The normal range of CPP in adults is 70–100 mmHg. CPP is compromised by a failure in autoregulation, the intrinsic capacity of blood vessels in the brain to regulate and maintain normal blood flow. When systemic arterial pressure falls, or a cerebral artery is partially or completely occluded, blood vessels in the brain dilate in a compensatory at-
History and Physical Findings

For the patient presenting with altered mental status, emergency evaluation is indicated. Once a patient has been determined to have altered consciousness, immediate assessment of airway, breathing, and circulation is required. Obtaining information regarding the patient’s previous medical history, current health status, and timeline for the current complaint from a significant other or caregiver is integral in uncovering the cause. When the patient is stabilized, a more detailed examination should be performed that includes assessment of skin color and temperature, cardiovascular examination for bradycardia and hypotension, and constitutional findings such as alterations in gastrointestinal function (incontinence, diarrhea, or vomiting) and genitourinary complaints (urinary retention or incontinence). Focused components of the neurologic examination include assessment of mental status (memory, concentration, orientation, mood, affect, speech, psychomotor activity, and the presence of delusions or hallucinations), cranial nerve function, and presence of weakness (Vollmer & Dacey, 1991).

Diagnostic Tests

The goal of the physical examination and diagnostic testing is to uncover the underlying cause of altered consciousness so that appropriate treatment can be offered. The differential diagnosis of altered mental status in patients with cancer is extensive and may include metabolic derangements; septicemia; hypoxia; and central nervous system effects, such as hemorrhage, encephalopathy, infection, or tumor (see Figure 2). Diagnostic tests should include a chemistry panel, complete blood count, antiepileptic drug medication level (if appropriate), oxygen saturation, and imaging of the brain with CT scan or magnetic resonance imaging (MRI) (Nolan, 2005). CT scans are quick and relatively inexpensive when compared to MRI and are a good test in an acute situation to evaluate for hydrocephalus or hemorrhage. However, MRI provides improved assessment of circulation, anatomy, and characteristics of a brain tumor. Figures 3–7 provide pregadolinium axial T1-weighted MRIs of causes of increased ICP. W.C.’s imaging revealed a subdural hematoma, resulting in increased ICP and altered consciousness.

Management

Increased ICP disturbs brain function by reducing cranial blood flow or causing brain herniation. The goals of treatment are to determine the cause of ICP and initiate treatment to reduce ICP so that cranial blood flow adequately oxygenates the brain and herniation is relieved (Youmans, 1982). In patients with subtle signs of increased ICP related to tumor, such as early morning headaches and focal neurologic symptoms, corticosteroids often are used to reduce cerebral edema. If the tumor is...
figure with associated mass effect, surgically debulking also may be required to reduce pressure.

Immediate treatment of acute, increased ICP is required to reverse or prevent cerebral herniation and to prevent death (DeAngelis, Gutin, Leibel, & Posner, 2002). Maintaining a clear airway is critical in patients who are acutely decompensating. Carbon dioxide retention causes cerebral vasodilation, which further raises ICP (Cairncross & Posner, 1981). Measures to control increased ICP include hyperventilation, head of bed elevation, corticosteroids, osmolar therapy, diuretics, ventricular drainage, neuromuscular blockade, and barbiturate therapy (DeAngelis et al., 2002; Dunn, 2002; Thapar et al., 2001). Hyperventilation decreases partial pressure of carbon dioxide, resulting in vasoconstriction and lowered ICP. Hyperventilation is the fastest method for lowering ICP, but its effect is short-lived as the body compensates for induced respiratory alkalosis by producing metabolic acidosis. Elevating the head of bed will lower ICP by improving venous flow. Corticosteroids are used to treat increased ICP by decreasing vasogenic edema. Mannitol is the most commonly used agent for osmolar therapy; it promotes the movement of water out of the brain tissue by creating an osmotic gradient between the blood and the portion of the brain with an intact blood-brain barrier. Diuretics such as furosemide lower ICP in the setting of cerebral edema. When hydrocephalus is present, placement of an intraventricular catheter for ICP monitoring is used for ICP control and drainage of CSF through a ventriculostomy. Neuromuscular blockade is used to control muscle activity, which can further increase ICP. Barbiturate therapy was used in the past to reduce persistent increased ICP resistant to other therapies. Barbiturates lower ICP by decreasing cerebral metabolism, which causes reduction of cerebral blood flow and ultimate lowering of ICP (Dunn, 2002). However, the use of barbiturates is associated with significant, prolonged effects, such as hypotension, and patients receiving them require intensive monitoring. Therefore, barbiturates are no longer used in this setting (Hodgkinson & Mahajan, 2000).

Nursing Implications

Oncology nurses equipped with knowledge and skill in neurologic emergencies can detect early signs and symptoms of increased ICP and favorably impact the outcome of such patients (Nolan, 2005). In patients with acute exacerbation of increased ICP, early intervention can prevent cerebral herniation and even death. Oncology nurses caring for patients with cancer at risk for increased ICP can perform and document a complete, concise, neurologic assessment and detect subtle neurologic changes that should lead to timely intervention and management. Other nursing responsibilities include maintaining the position of the patient’s head elevated at a 30°–45° angle. That technique improves jugular venous outflow and lowers ICP. CSF drainage must be monitored closely if an intraventricular catheter is used. Although cerebral edema is managed with drugs like mannitol, dexamethasone, and diuretics, nurses assess patients for side effects, which include hypersensitivity, dehydration, electrolyte imbalance, fluid retention, and hypotension. Increased ICP also can precipitate seizure activity. For patients who have seizures because of increased ICP associated with brain tumors, aggressive management with anticonvulsants is the optimal intervention. Oncology nurses should assess the needs of patients’ families for education aimed at helping them understand the disease process and its consequences. Oncology nurses also can help family members cope by encouraging them to express feelings, listening actively to their concerns of fear and uncertainty and providing emotional support.

Patient Follow-Up

W.C. underwent emergency evaluation, and imaging revealed a large subdural hematoma. He was given mannitol and taken emergently to the operating room for evacuation of the hematoma. After surgery, he remained in the intensive care unit for three days and then required 14 days of inpatient rehabilitation because of postoperative deficits of weakness and gait imbalance. One month after surgery, he was able to ambulate independently and continue with chemotherapy. Anticoagulation was not continued because of the intracranial hemorrhage.

Summary

Brain tumors and neurologic complications of cancer are occurring with increasing frequency, partly because diagnostic procedures have improved and more brain tumors are being identified. Increased ICP is one of the most frightening and potentially lethal complications associated with brain tumor histologies. The goals of management are to prevent the occurrence of this event and maintain patient safety.

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Figure 6. Herniation of Midline Structures

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Figure 7. Increased Intracranial Pressure and Herniation

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References


