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Unfortunately, when laparoscopy is associated, this is uncomfortable for the patient.

In summary, to minimize the risk of severe acute dilutional hyponatremia and intravascular overload, we manage the patients undergoing endoscopic YAG laser uterine surgery as follows. Intravenous fluids are given as sparingly as possible. An artificial menopause is induced several weeks before surgery with GnRH implant or lyn
estrenol to decrease the liquid absorption by the endometrium and reduce the uterine volume. Whenever possible, regional anesthesia is preferred. When a laparoscopy is associated with the hysteroscopic surgery, as much liquid as possible is reaspirated through the laparoscope and if not, by culdocentesis. The absorption of fluid is estimated and serum sodium is monitored when fluid absorption exceeds 1,500 ml. In high-risk patients, insertion of a central venous pressure catheter is considered.

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Cardiorespiratory Arrest Following Initiation of Cranial Irradiation for Treatment of a Brain Stem Tumor

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Central nervous system (CNS) radiotherapy is frequently employed in the treatment of intracranial tumors. 1 Although rare, adverse reactions to CNS radiotherapy have been reported. Pochely described radiation-induced encephalopathy that occurred weeks to months following therapy. 2 Acute herniation has also been described in a special subset of patients with meningeal leukemia. 3 We report a case of acute neurologic deterioration following radiotherapy in a patient with a malignant brain stem tumor.

REPORT OF A CASE

The patient was a 5-yr-old, 20 kg, white girl with a 2-wk history of headaches, right sided extremity and facial weakness, decreased sensation in the right arm, gait ataxia, and slurred speech. Computed tomography (CT) and magnetic resonance imagery (MRI) brain scans demonstrated a large left pontomesencephalic mass without hydrocephalus. The patient was taken to the operating room for a stereotactic biopsy of her brain stem lesion. The procedure was uneventful, and the permanent pathologic sections revealed undifferentiated, primitive neuroepithelial medial tumor. Four days later the patient was discharged from the hospital, at which time she was receiving dexamethasone 4 mg four times a day.

Three days later the patient developed a worsening of her right hemiparesis and increased lethargy. CT scan revealed a new rim of edema surrounding the tumor without significant hydrocephalus. In an effort to shrink the lesion, a palliative course of brain stem radiotherapy was planned. Radiotherapy treatment was facilitated by the use of 40 mg meperidine, 20 mg phenergan, and 20 mg chlorpromazine given by intramuscular injection. The procedure was uneventful apart from posttreatment sedation of approximately 8 h. Because of this and the need to position the patient prone, general anesthesia was planned for the second treatment. Anesthesia was induced with 125 mg sodium thiopental in divided doses, 1 mg midazolam, and 4 µg sufentanil. Vecuronium (1 mg) was used to facilitate intubation. Following tracheal intubation and patient positioning, controlled hyperventilation was instituted with 100% oxygen to achieve an end-tidal carbon dioxide level (ETCO2) of 30 mmHg. After the 5- to 10-min radiotherapy session, 10 mg edrophonium with 0.5 mg atropine was given to reverse the effects of the muscle relaxant. Naloxone (100 µg) was administered slowly to antagonize the sufentanil. The trachea was extubated smoothly after patient arousal, and the patient was sent to the postanesthesia care unit (PACU) drowsy and with stable vital signs. Following 2 h of observation in the PACU, the patient’s level of consciousness had recovered to her pretreatment level and she was discharged to the ward. Two hours after her return to the ward she was found lying on the floor, unresponsive, apneic, and with vomiting draining from her mouth. No signs of focal motor or generalized seizures were noted. Cardiopulmonary resuscitation and tracheal intubation were successfully accomplished. Neurologic examination revealed pupils that were equal and reacted to light. All reflexes were present, and the patient was

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able to move all extremities. CT scan of the brain showed no change in the size of the tumor or extent of edema compared with that in the previous scan and no evidence of brain stem hemorrhage or hydrocephalus. The patient was transferred to the intensive care unit where hyperventilation was continued. A ventricular catheter was inserted and revealed low intracranial pressure (<10 mmHg). Cerebrospinal fluid (CSF) examination showed no evidence of infection. After several days the patient showed progressive neurologic improvement. She was gradually weaned from ventilatory support over the next week, and the trachea was extubated 12 days after her respiratory arrest. Neurologic examination performed 2 wk after her arrest demonstrated a complete recovery to her prearrest neurologic status.

DISCUSSION

Unexpected postoperative cardiopulmonary arrest is a catastrophic event. The most likely causes of this event in this patient include residual effects of anesthesia, acute electrolyte or metabolic disturbance, seizures, obstructive hydrocephalus, bleeding into the tumor, or acute cerebral edema.

The presentation of this child prior to radiation therapy with symptoms of lethargy and signs of brain stem involvement, together with CT findings of a large pontomesencephalic tumor with surrounding edema, suggested the presence of increased local posterior fossa intracranial pressure and the potential for developing generalized intracranial hypertension if the cerebral aqueduct became occluded. Intravenous anesthesia is usually administered in this situation. However, in this instance the anesthetic used was chosen to ensure a stable intracranial pressure (ICP) and a motionless patient during the treatment period; the drugs employed were used because their effects are rapidly reversed and because these drugs would minimize any anesthetic contribution to an increasing ICP. The patient was observed for 2 h in the PACU, at which time the nurse, anesthesiologist, and the patient's mother agreed that she was at her preanesthetic level of consciousness. This course of events makes an anesthetic-induced complication unlikely. A seizure was thought to be an unlikely cause of the cardiac arrest because the tumor was infratentorial and there were no previous signs of seizure activity. Serum electrolytes and glucose determination failed to reveal any abnormality. CT excluded the diagnosis of tumor necrosis or bleeding into the tumor. Furthermore, there was no evidence of sudden hydrocephalus. Therefore, the question of radiotherapy-induced acute cerebral edema was raised. Indeed, the pretreatment CT scan showed a new area of edema around the tumor. With a tumor localized to the brain stem, even small amounts of local tissue swelling could cause pressure on vital centers.

Whether CNS radiotherapy may worsen cerebral edema is not known. A review of the literature revealed a single case report of five children with CNS leukemia who had undergone radiotherapy following intrathecal chemotherapy. The authors of this report postulated that the widespread diffuse involvement of CNS leukemia may put these patients at higher risk for cerebral edema following destruction of tumor cells. The time course of initial cell membrane damage and the concomitant edema is not well established but thought to occur within the first week of treatment. In the case reported here the initial treatment was 60 h prior to the arrest. This time course suggests a possible relationship between the radiotherapy and the subsequent arrest seen in this patient. This case report is presented to draw attention to potential postradiotherapy treatment problems that may be encountered in this small subset of children. We recommend that such patients require prolonged monitoring of respiration after radiation.

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