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Cardiac Arrest Following Rapid Drainage of Cerebrospinal Fluid in a Patient with Hydrocephalus

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Cardiac arrhythmias occasionally occur in patients with intracranial disease who have no intrinsic heart disease. Sudden changes in intracranial pressure may produce movement of the brain stem and hypothalamus and further contribute to the appearance of cardiac rhythm disturbances. We present the case of a patient with a tumor of the hypothalamus and hydrocephalus in whom severe cardiac dysrhythmia and cardiac arrest developed following rapid decompression of elevated intracranial pressure.

REPORT OF A CASE

An 18-year-old, 55-kg woman was admitted to the hospital with a chief complaint of diplopia during right lateral gaze. Although she had had a headache lasting a month after spinal anesthesia administered for childbirth six weeks previously, she denied headache and other symptoms of intracranial hypertension at this admission.

Physical examination disclosed right sixth cranial nerve palsy and early papilledema. A computed tomographic (CT) scan revealed numerous calcifications in the hypothalamus and bilateral ventricular enlargement. By pneumoencephalography, during which cardiac arrhythmias did not occur, a mass was found within the hypothalamus, obliterating the foramen of Monroe. Radiograms of the skull showed that small amounts of residual intraventricular air were present following pneumoencephalography.

Bilateral ventriculoperitoneal shunting was scheduled as treatment for hydroencephalus. Dexamethasone, 4 mg, was given orally every six hours on the day before operation. No premedication was given, and anesthesia was planned to minimize the risk of intracranial hypertension and to avoid nitrous oxide because of the presence of intraventricular air. Fentanyl, 0.2 mg, iv, was given in divided doses while the patient breathed 100 per cent oxygen and was asked to hyperventilate. Thiopental, 250 mg, and pancuronium, 8 mg, were given iv. Ventilation was then controlled and halothane, 0.5–1 per cent, was added to the inspired mixture. When paralysis was adequate, as judged by response to peripheral nerve stimulation, the trachea was intubated and anesthesia was continued with halothane, 0.8 per cent, in oxygen. Heart rate and arterial blood pressure were 85 beats/min and 120/70 torr, respectively, prior to induction of anesthesia; they increased to 95 beats/min and 130/85 torr following induction.

About 90 minutes later, a ventricular catheter was inserted, after which an unmeasured quantity of cerebrospinal fluid immediately escaped under high pressure. Ventricular tachycardia suddenly occurred, which rapidly progressed to ventricular fibrillation. Lidocaine, 100 mg, was given; a precordial thump was followed by external cardiac massage. After approximately 15–20 compressions of the chest, a sinus tachycardia at 170 beats/min appeared, interspersed with multifocal premature ventricular contractions. Arterial blood pressure was 160/110 torr. Lidocaine, 150 mg, and propranolol, 1 mg, were given iv over the succeeding 10 min in divided doses. Heart rate and arterial blood pressure decreased to 90 beats/min and 120/80 torr, respectively, and the premature ventricular contractions disappeared. Nitrous oxide, 60 per cent, was added to the inspired gas mixture, and additional doses of pancuronium and thiopental were given iv as anesthesia and operation continued uneventfully. Cardiac dysrhythmia did not recur, and no other sequelae of this event developed.

DISCUSSION

Electrocardiographic abnormalities associated with central nervous system disorders are well known. Conditions associated with increased intracranial pressure, such as subarachnoid hemorrhage, intracranial space-occupying conditions, and meningitis, have been linked to ST-segment and T-wave abnormalities.[1–3] In addition, disturbances of cardiac rhythm have been reported, usually in association with subarachnoid hemorrhage.[4–7] The etiology of arrhythmias seen with increased intracranial pressure remains speculative, but most investigators believe that movement of the brain stem and hypothalamus and changes in autonomic outflow are involved.[4,8–11]

In obstructed hydrocephalus, the location of decompression is important in determining the physiologic changes that might occur. Removal of spinal cerebrospinal fluid (CSF) may cause a downward herniation of the brain and pressure on the lower brain stem to produce apnea. Conversely, removal of ventricular CSF could cause a cephalad movement of the brain and pressure on the hypothalamus, with production of cardiac arrhythmia. Acute decompression of experimentally induced intracranial hypertension has been shown to provoke cardiac arrhythmias in anesthetized dogs.[9] One case of cardio-

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vascular collapse occurring after rapid ventricular decompression in a 6-month-old child with hydrocephalus has been reported.[12] The authors presumed cardiac arrest, although this was not confirmed by electrocardiogram. External cardiac massage for a minute failed to restore a palpable pulse. Injection of 40 ml saline solution into the ventricles through the decompression tube was associated with the reappearance of audible heart tones and spontaneous ventilation. The authors attributed successful resuscitation to the injection of saline solution into the ventricular system.

Our patient suffered cardiac arrest following rapid drainage of cerebrospinal fluid for treatment of hydrocephalus; to our knowledge, this represents the first electrocardiographically documented episode to be reported. A rostral shift of the brain might have placed further pressure on the hypothalamus, an area recognized as a sympathetic discharge center. Ventricular tachycardia and fibrillation resulted from this sudden shift. Treatment with intravenous lidocaine and propranolol and external cardiac massage was successful; no permanent damage resulted.

The time of decompression of elevated intracranial pressure may be hazardous. Arrhythmia might be produced by movement of brain stem structures and changes in autonomic outflow. We suggest that ventricular pressure should be reduced gradually, in a controlled fashion, while cardiovascular dynamics and rhythm are monitored for changes. Should arrhythmia develop, CSF drainage should be halted. If arrhythmia persists or worsens despite conventional pharmacologic treatment, then the collected CSF or saline solution should be cautiously infused into the ventricular catheter.

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