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Intraoperative Subdural Tension Pneumocephalus Arising after Opening of the Dura

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Perioperative tension pneumocephalus was first described by Ectors[1] in 1962 and has been described many times since.[2–11] It has, however, never been reported as occurring intraoperatively in the subdural space when the dura is open. We describe two cases in which subdural tension pneumocephalus occurred during craniotomy after dural opening. Both of these presented as acute, severe brain swelling of unknown etiology.

REPORT OF TWO CASES

Case 1. A previously healthy 63-yr-old, 71-kg woman presented with a 6-month history of increasingly frequent frontal headaches. Clinical examination revealed mild bilateral papilledema and an ataxic gait. A computerized tomography (CT) scan demonstrated a right-sided lesion, enhanced with contrast, that was adjacent to and obstructing the third ventricle. A magnetic resonance imaging (MRI) scan suggested a vascular malformation.

The patient was scheduled for a bifrontal craniotomy in the supine position. Preoperatively, dexamethasone (Decadron® 4 mg four times per day), ranitidine (150 mg two times per day), and phenytoin (300 mg/day) were given. All laboratory data were within normal limits. Blood pressure was 130/80 mmHg. No additional preanesthetic medication was prescribed.

Anesthesia was induced with thiopental 300 mg and fentanyl 400 μg, and the trachea was intubated after vecuronium 10 mg. Anesthesia was maintained with N_2O 70% in O_2 and the arterial hemoglobin O_2 saturation (Sp_{O_2}) kept between 96–98%. Throughout surgery, core body temperature, intraarterial blood pressure, central venous pressure, end-tidal CO_2 partial pressure (PET_{CO_2}), neuromuscular blockade, Sp_{O_2}, and breath sounds were continuously monitored. Ventilation was controlled to maintain an PET_{CO_2} of 27 mmHg.

The head was placed in the neutral position and immobilized in Mayfield pins. Mannitol 100 g and furosemide 20 mg were administered. A diuresis of 2,100 ml had occurred by the time the dura was opened (90 min after diuretics). Prior to incision, an additional 300 μg fentanyl was administered. Trimetaphan was used to maintain the systolic blood pressure between 100 and 120 mmHg intraoperatively. A bifrontal craniotomy was performed, and the dura was opened over the right hemisphere adjacent to the falx cerebri.

Initially the brain was quite shrunken, but 30 min later it began to swell, and increased venous bleeding developed. No change in physiologic parameters had occurred, and the breathing circuit was not obstructed. A surgical cause of the swelling was suggested; however, on palpating the intact dura over the left hemisphere, the surgeon believed that "a subdural hemorrhage was unlikely."

Surgery proceeded, and the right hemisphere was gently retracted to gain access to the lesion. As the sucker was applied along the right side of the falx cerebri to evacuate clot, air under pressure was suddenly released through an aperture in the falx. This was followed by a prompt resolution of the swelling and bleeding. It appeared that subdural air had collected over the left hemisphere and that blood had sealed its point of entry.

No further complication was encountered, and a cavernous hemangioma was excised. The patient made an uneventful recovery.

Case 2. A 26-yr-old, 64-kg woman presented with a 3-week history of right-sided weakness and mild motor dysphasia. Clinical examination revealed mild weakness and hyperreflexia in both the right upper and right lower limbs and early bilateral papilledema. A MRI scan identified multiple areas of altered signal intensity throughout the left cerebral hemisphere, suggestive of metastases. No primary lesion could be identified, and the patient was scheduled for an open biopsy through a left parietal craniotomy in the semi-sitting position. Laboratory data were within normal limits. Dexamethasone (Decadron® 4 mg four times per day), ranitidine (150 mg two times per day), and phenytoin (300 mg/day) were given preoperatively.

The patient received temazepam 20 mg orally. After the monitoring (as in Case 1) was established, anesthesia was induced with thiopental 300 mg and fentanyl 500 μg, and after vecuronium 10 mg was given, the trachea was intubated. Anesthesia was maintained with 70% N_2O in O_2 while the Sp_{O_2} was kept between 95–97% and the lungs ventilated to an PET_{CO_2} of 26 mmHg.

The head was immobilized, brow uppermost, in Mayfield pins and elevated to 20 cm above the heart. At the same time, mannitol 75 g and furosemide 20 mg were administered, and 2,500 ml urine was produced by the time the dura was opened (100 min after diuretics).

Prior to dural incision the brain was noted to be shrunken. The dura was opened over the left parietal lobe adjacent to the falx cerebri. Almost immediately the brain began to protrude into the craniotomy and by 15 min had swollen to an alarming degree. A rapid appraisal of the anesthetic technique (as for the previous case) was unable to

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identify a cause. A contralateral subdural hematoma was suspected; however, extending the craniotomy did not disclose a hematoma.

At this stage, subdural tension pneumocephalus was considered. N_2O was discontinued and ventilation continued with 1% isoflurane in O_2. Despite its rapid onset, the swelling abated, to a point where a suction catheter could be passed anteriorly along the falx cerebri to the frontal subdural compartment. The swelling lessened further, but a return to the initial state of relaxation was not achieved.

Surgery continued, and a frozen section revealed a high-grade glioma. Subtotal radical resection was performed along the left cingulate gyrus.

Upon completion of surgery, but prior to termination of anesthesia, a CT scan was performed and revealed a 7 × 1 cm collection of frontal subdural air.

It was decided to treat the intracranial air expectantly. The patient readily awakened from anesthesia and the trachea was extubated. No change in her preoperative condition was evident, and her postoperative course was uneventful.

Discussion

Tension pneumocephalus always relates to a sealed, gas-filled compartment in which the volume increases or intracranial compliance is compromised. In the latter situation, if the volume of brain, cerebrospinal fluid (CSF), or cerebral blood subsequently increases, then intracranial pressure (ICP) may increase to a critical degree. The etiology of this air can be classified as follows:

1. Closure of an air-filled compartment: *e.g.*, dural closure over a tumor resection cavity not filled with saline[2]
2. Air forced intracranially
   A. Negative ICP: *e.g.*, ventricular shunts[3] and ventricular catheters,[4] and craniotomy for patients in the sitting position[5]
   B. Positive extracranial pressure, *e.g.*, pneumocephalograms[6]

The volume of a sealed gas-filled compartment may increase in size, either by diffusion of N_2O or by warming of room temperature air to body temperature. Raggio[12] has demonstrated that the effect of warming will cause an increase in volume of only 4%. The effect of N_2O depends on the inspired concentration and the composition of the trapped gas. N_2O will diffuse into an air-filled cavity 34 times faster than N_2 diffuses out. This means that 50% N_2O, at a maximum, will lead to a doubling of the volume, and 75% will quadruple it (assuming a constant pressure). Saidman and Eger[6] showed that, in healthy dogs after the injection of air into the cisterna magna and in humans after pneumoencephalogram, exposure to 75% N_2O resulted in increased ICP, with the peak pressure occurring at 10 min. The time required for pressure to decrease after discontinuation of N_2O was similar.

Most cases of symptomatic pneumocephalus occur in the postoperative period, even when N_2O has not been used or terminated prior to dural closure.[7] The retained air compromises cerebral compliance, and the increase in brain and blood volume that occurs after surgery may lead to symptomatic increases in ICP.[8]

These are the first reported cases in which intraoperative subdural tension pneumocephalus occurred with the dura open. We were able to find only three other reports of cases in which tension pneumocephalus was recognized during surgery. Artru[9] and Thiagarajah *et al.*[10] reported subdural tension pneumocephalus arising immediately after dural closure. Drummond[11] reported a case of sudden brain swelling occurring 6 h after air was accidentally allowed to enter the ventricles through a ventriculostomy needle. In this case the problem was probably the result of acute cerebellar swelling in the presence of impaired cerebral compliance, rather than N_2O diffusion into the air-containing space.

Both of our cases involved the entry of air into the subdural space and blockage of its egress. The generation of the necessary negative subdural pressure presumably was the product of reduced brain volume and the tethering of the dura to the inner table of the skull by sinuses and veins. After dural opening, the entry of air allowed the brain to fall away from the uppermost skull, leaving an air pocket.

In Case 1, the air was entrained across the aperture in the falx. These apertures are common particularly in the narrow anterior portion of the falx.[13] Presumably, the aperture then was sealed by a blood clot. The subsequent diffusion of N_2O into the air-containing space increased its pressure to a condition mimicking brain swelling. Removal of the clot allowed for diagnosis and decompression.

In Case 1, the air entered the subdural space through the craniotomy and accumulated in the uppermost part of the skull. The exact mechanism for the prevention of egress of the air is unclear. We hypothesize that while a negative subdural pressure exists, the nondependent brain is held suspended from the dura rather like the lung from its apical pleura. When the dura was opened, air entered and thereby lead to a loss of subatmospheric pressure that allowed the brain to settle into the inferior cranial vault. Since the brain volume is fixed (over such a short period of time), this decrease in anteroposterior length must lead to an increase in lateral width, and so this settling movement then occluded the craniotomy opening. That would correlate with the sudden condition mimicking brain swelling that occurred within seconds of dural opening, and would confirm the subsequent slower deterioration as due to N_2O diffusion. The improvement with discontinuation of N_2O relates to its reabsorption, but complete resolution was not achieved due to the weight of the brain.

If brain swelling occurs, after dural opening, when conditions are present such that a subatmospheric subdural pressure may have been present, then a diagnosis of tension pneumocephalus should be considered.

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Thrombocytopenia and Cocaine Abuse

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This report describes a case of severe thrombocytopenia associated with cocaine abuse. Anesthetists are cautioned to rule out thrombocytopenia in patients who are known substance abusers and to consider illicit drug use in patients with unexplained low platelet counts. The etiology and management of autoimmune thrombocytopenia in drug abusers is discussed.

CASE REPORT

A 26-yr-old, approximately 70-kg man presented to hospital 12 h after sustaining a blow to the head. He was known to chronically use cannabis and cocaine. Upon examination, the man was found to be stuporous; he was able to open his eyes to command but unable to respond to verbal questions. His blood pressure was 140/80 mmHg, heart rate 70 beats per min, and respiratory rate 20 breaths per min. There was a contusion over the left occipital area; his pupils were equal and reactive but showed blurring of the optic discs; and his left arm was notably weak on flexion. Multiple skin puncture sites were noted along his forearms. Initial blood laboratory analysis was as follows: platelets 17 · 10^9/l (normal value 150–400 · 10^9/l), hemoglobin 124 g/l, white blood cells 12.6 · 10^9/l, neutrophils 81%, lymphocytes 16%, monocytes 3%, reticulocytes 130 · 10^9/l (normal value 40–100 · 10^9/l).

A traumatic intracerebral hematoma was suspected, and 200 ml mannitol 20% was administered. The patient was transferred to our neurosurgical institute where a computerized tomography scan revealed a large right frontal hematoma, contusion of the right frontal lobe, and intraventricular hemorrhage with a marked midline shift.

The patient was given another 200 ml mannitol 20% and was transferred to the operating room, where craniotomy and evacuation of the right frontal hematoma was performed. The patient was anesthetized with isoflurane, nitrous oxide, and fentanyl. The 3-h procedure was tolerated without incident, but the surgeons noted excessive oozing from the incision. Twelve units of pooled platelets were administered prior to transfer to the neurosurgical intensive care unit. Twenty hours postoperatively, the patient regained consciousness and the trachea was extubated. Unfortunately, he sustained a marked functional neurologic deficit and was transferred to a rehabilitation center on day 30 of admission.

His platelet profile during his hospital admission was as follows: immediately posttransfusion, 185 · 10^9/l; day 1 postsurgery, 65 · 10^9/l; day 14, 855 · 10^9/l; and day 26, 383 · 10^9/l. Subsequent blood analysis showed no evidence of a consumptive coagulopathy: prothrombin time, partial thromboplastin time, plasma fibrinogen, and thrombin time were normal. The Venereal Disease Research Laboratory (VDRL) syphilis test and hepatitis screen were negative. Immunoassays for antiplatelet antibodies and circulating immune complexes were not performed. Human immunodeficiency virus antibody testing was not performed because of lack of informed patient consent.

DISCUSSION

The medical and anesthetic considerations of cocaine abuse have been reviewed previously.[1,2,†] Thrombocy-

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