Delayed emergence due to intracranial hemorrhage after middle ear surgery: A case report

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Abstract
A 63-year-old male patient had delayed emergence from general anesthesia after a micro-resection of the left temporal bone. After a complete physical examination of the nervous system, an emergent brain computed tomography (CT) was arranged, revealing left frontotemporal and parietal subdural hematomas and subfalcine herniation. Intracranial hemorrhage is an unpredictable and rare complication after ear surgery that physicians need to be aware of.

Keywords: intracranial hemorrhage; delayed emergence from general anesthesia; temporal bone

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Introduction
Delayed awakening from anesthesia is a common complication. The time taken to emerge to full consciousness is affected by patient factors, anesthetic factors, the duration of surgery, and painful stimulation[1]. There are many reasons that account for delayed awakening. Residual sedation from general anesthetics or opioids used during anesthesia is the most frequent cause of delayed awakening in the PACU. The other reasons, non-pharmacologic causes, including metabolic and neurologic causes, may have serious sequelae[1]. In addition, hypothermia, advanced age, and renal disease may contribute to prolonged recovery from anesthesia due to the increased sensitivity to or slowed elimination of the administered medications[2]; however, delayed emergence can be secondary to unusual causes and present a diagnostic dilemma for reasons including drug interactions, serotonin syndrome, post-operative delirium, central anticholinergic syndrome, psychiatric disorders, narcolepsy/sleep paralysis, surgical complications, and total spinal anesthesia[3].

Neurologic causes of delayed awakening include intracranial hemorrhage, cerebral ischemia, and medullary compression. Intracranial hemorrhage is a common neurologic emergency. In most cases, intracranial hemorrhage is due to the effects of longstanding hypertension on small vessels. The other causes of intracranial hemorrhage include blood vessel abnormalities, injuries, tumors, inflammation of blood vessels, bleeding disorders, and use of anticoagulants[4]. Surgical complications are also uncommon reasons for intracranial hemorrhage[3]. Peri-operative intracranial hemorrhage is an important cause of morbidity and mortality, particularly following cardiac, neurologic, and carotid surgery[5]. In fact, greater than one-half of the patients who have a large hemorrhage post-operatively die within a few days. Early mortality may result from delayed recognition and diagnosis on surgical services, cerebral edema, and intracranial hypertension. The overall mortality for hemorrhagic strokes is higher than ischemic stroke and the risk of mortality is up to 52% in the first 2 days[6]. Thus, a quick work-up for delayed emergence is very important because the differential diagnosis, including causes, is significantly associated with morbidity and mortality. Early detection and correct interpretation of the typical bleeding pattern might help to avoid further aggravation of symptoms[7]. The diagnosis of intracranial hemorrhage is based on symptoms,
physical examination, and imaging examination, but may be challenging in the context of the anesthesia state.

Case report

The patient was a 63-year-old male (weight = 67 kg) who presented with the diagnosis of a left temporal bone tumor. He was scheduled for microsurgical resection of the temporal bone. No remarkable medical history was noted pre-operatively. The laboratory values were within normal ranges. Routine blood coagulation tests were normal.

Anesthesia was induced with etomidate (0.3 mg/kg), sufentanil (0.5 μg/kg), and atracurium (0.6 mg/kg). After endotracheal intubation, he was placed in the dorsal position with head tilted slightly to right side. Anesthesia was maintained with sevoflurane and remifentanil (0.15 μg/kg/min). Controlled hypotension was established with the systolic blood pressure in the range of 80 ~ 130 mmHg and a heart rate of 55 ~ 70 bpm intraoperatively. The PETCO$_2$ was maintained at 35 ~ 45 mmHg. All anesthetics were discontinued at the completion of surgery.

The surgery lasted for 360 min. The patient underwent resection of the left temporal bone and tympanoplasty using the Fisch technique under an operating microscope. The total amount of intraoperative blood loss was approximately 400 ml. Twenty minutes after discontinuation of the anesthetics, the end-tidal sevoflurane concentration was almost 0. His spontaneous respirations were regular and the muscle relaxant was reversed with 30 μg/kg of prostigmine; however, the patient did not respond to verbal commands. A painful stimulus resulted in a withdrawal response without opening the eyes. The pupils were isocoric with a diameter of 4 mm; the pupillary light reflexes were decreased bilaterally. Tracheal extubation was performed 10 min later because the patient was not tolerating the tracheal tube. The patient was transferred to PACU for further recovery.

Thirty minutes later, the vital signs remained normal and stable, but the patient remained unconscious. Delayed recovery from anesthesia was considered and naloxone (0.2 mg) was injected, but no improvement was observed. Arterial blood gas analysis was normal and blood glucose was within normal range. There were no electrolyte disturbances. The pharyngeal temperature was 37°C.

A neurologist was consulted. The pupils were equal (to 5 mm), the pupillary light reflexes were absent bilaterally, and the Babinski sign was positive bilaterally. After a complete examination of the nervous system, an emergent brain computed tomography (CT) was arranged 60 min post-operatively, revealing left frontotemporal and parietal subdural hematomas and subfalcine herniation (Fig.1). The patient promptly underwent a hematoma evacuation and decompressive craniotomy. Several bleeding points were identified that resulted from rupture of small dural vessels in which hemostasis had been previously achieved with compression and electric coagulation. Unfortunately, the patient died from encephal edema 2 days later.

Computed tomography (CT): left frontotemporal and parietal subdural hematomas and subfalcine herniation (Fig.1).

Discussion

Even after prolonged surgery and anesthesia, a response to stimulation after 60-90 min should occur. In the present case, we first reviewed the anesthetic chart, then naloxone, a specific antagonist against opiate receptors, was administered. The end-tidal sevoflurane concentration and TOF-Watch did not support the residual effect of anesthetics or the muscle relaxant. The arterial blood gas and serum electrolyte analysis helped us exclude metabolic factors. Intra-operative hypoxemia or ischemia, hypertension or hypotension, which may result in brain destruction, did not exist in this case.
After conservative treatment in the PACU for 60 min, an emergent brain CT revealed an intracranial hemorrhage.

Peri-operative intracranial hemorrhage often occurs in cardiac, neurosurgical, and carotid artery surgeries[8]. Other case reports involving intracranial hemorrhage include complicated cervical or lumbar surgery[9], a left medial wall orbital decompression for thyroid-related optic neuropathy[10], and thrombolytic therapy[11]. Risk factors for non-cardiac and non-vascular surgeries include the type of surgery and surgical site, age > 70 y, a history of coronary artery disease, chronic congestive heart failure, chronic kidney disease, cerebrovascular disease, diabetes mellitus, or hypertension, a peri-operative bleeding disorder, a pre-operative abnormal ECG, intra-operative hypotension, and a blood transfusion[12,13]. Several cases of cerebellar hemorrhage have been reported following spine surgery, which may have occurred due to the change in cerebral fluid pressure secondary to a cerebrospinal fluid leak[14]. Intracranial hemorrhage is an unpredictable and rare complication following ear surgery[15]. Gürbüz et al.[16] reported a case of subdural hematoma occurring as an extremely rare and life-threatening complication of cochlear implantation. In the present case, none of these risk factors existed and there was no cerebrospinal fluid leakage intra-operatively, which is another reason that intracranial hemorrhage was not considered initially.

The diagnosis of intracranial hemorrhage is difficult when the patient is under anesthesia. The patient might not exhibit a severe headache, nausea, or vomiting. The pupils may become abnormally large or small as a result of intracranial lesions. A change in pupil size cannot be observed by anesthesiologist intra-operatively during ear surgery. In addition, use of anticholinergic drugs, such as atropine or prostigmine, impairs the ability to note changes in pupil size. The Babinski sign is a well-known indicator of upper motor neuron dysfunction; the Babinski sign is routinely used to determine whether or not a lesion of the pyramidal tract exists and can be regarded as a tool to help determine lesion localization[17]. Every physician should perform this neurologic examination to expedite the differential diagnosis of delayed emergence; however, a positive Babinski sign can appear during normal sleep or recovery. The mechanism is not clear, but may result from differential recovery rates of various parts of the central nervous system following anesthesia[18]. These neurologic abnormalities in the recovery period are extremely difficult to identify. CT without contrast is the gold standard for ruling out a stroke. Other modalities, such as CT with contrast or magnetic resonance imaging, may be used if enhancement is needed[19]. Surgery in the middle ear always involves the skull base; however, most otolaryngologists concentrate on the facial nerve and hearing protection. Abnormal intracranial hypertension may be the only sign of intracranial hemorrhage under general anesthesia, and certainly captures the neurosurgeon’s attention[20]. In our case, this important sign was neglected by the otolaryngologist and anesthetist. The otolaryngologist noted significant hypertensive dural distention after temporal bone-flap elevation. Rather than investigate the cause of the hypertensive dural distention, the otolaryngologist ordered mannitol to reduce the intracranial pressure. The hypertensive dural distention was also dismissed by the anesthetist since the vital signs were stable. The heart rate was 55–70 bpm throughout the surgical procedure, severe bradycardia did not occur. Controlled hypotension during surgery obscured the hypertension which should accompany intracranial hypertension. Even in the PACU, the systolic blood pressure was not > 140 mmHg.

The outcome after peri-operative intracranial hemorrhage is usually devastating. The acute subdural hematoma mortality rate is significantly less if clots are removed within the first 2 to 4 hours after injury[21]. Prompt diagnosis and evacuation of hematomas are critical for patients with acute subdural hematoma. Monitoring electroencephalographic findings, cerebral regional oxygen saturation levels, intracranial pressure, and the bispectral index may help us recognize this devastating complication in a timely fashion[22,23]. In recent years, specific neurologic monitoring techniques that can be used intra-operatively, including near-infrared spectroscopy and transcranial Doppler ultrasonography, also provide a continuous quantitative signal of the physiologic variable most related to injury and amenable to intervention[24,25]. In consideration of costs and other reasons, these techniques are usually performed during cardiac and cerebral surgeries.

In summary, this case highlights the importance of promptly establishing a differential diagnosis and cause analysis in the presence of delayed awakening. Peri-operative intracranial
hence prompt detection and diagnosis is essential. Physicians should be aware of the possibility of intracranial hemorrhage after ear surgery.

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Competing Interests
Mingfeng Liao, Yilin Zhao, Yi Zhang, Xueren Wang, Ailin Luo, and Xiaohui Chi all deny any competing interests.

References: