Delayed Emergence from Anesthesia Resulting from Cerebellar Hemorrhage During Cervical Spine Surgery

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Cerebellar hemorrhage is an unpredictable complication of spinal surgery. We encountered a case of cerebellar hemorrhage presenting with delayed emergence from anesthesia and hemiplegia after resection of an intradural extramedullar tumor from the cervical spine. Postoperative brain computed tomography revealed hematoma in the cerebellar vermis and right cerebellar hemisphere. The patient made a gradual

erebellar hemorrhage is an unpredictable complication of spine surgery. Only a few case reports of this rare complication have been published (1-4). The onset of neurological disturbance has been reported more than 10 h after surgery. According to previous reports (1-4), patients were neurologically intact on awakening from anesthesia and anesthesiologists therefore may not have considered the possible the development of cerebellar hemorrhage during spine surgery. The mechanism of such hemorrhage in spine surgery remains unclear but loss of cerebrospinal fluid (CSF) seems important in the pathogenesis of this complication (3,4). The literature contains no cases in which cerebellar hemorrhage became apparent while diagnosing delayed emergence from anesthesia. We encountered a case of cerebellar hemorrhage during cervical spine surgery presenting with delayed emergence from anesthesia. The complication was suspected to be the result of transient loss of CSF.

Case Report

A 74-yr-old female was scheduled to undergo resection of an intradural extramedullar tumor of the cervical spine.

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recovery with conservative treatment. Although the mechanism of cerebellar hemorrhage remains speculative, loss of cerebrospinal fluid may play an important role. Cerebellar hemorrhage must therefore be considered in patients with unexplained neurological deterioration or disturbance on emergence from anesthesia after spinal surgery.

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The tumor was located at the level of C2 and compressed the spinal cord from the right side. Other than atrial fibrillation, no remarkable medical history was noted preoperatively. Anticoagulants had not been administered and no bleeding tendency was clinically observed before surgery. Anesthesia was induced with propofol 100 mg and fentanyl 0.1 mg IV. Nasotracheal intubation was facilitated with vecuronium bromide 10 mg under direct laryngoscopy. Anesthesia was maintained with propofol 6-8 mg \cdot kg⁻¹ \cdot h⁻¹ and fentanyl 1–2 μ g \cdot kg⁻¹ \cdot h⁻¹, and bispectral index (BIS) was monitored to allow the dose of propofol to be adjusted. After induction of anesthesia, the left radial artery was cannulated to enable continuous monitoring of arterial blood pressure. Systolic blood pressure was controlled during anesthesia and never exceeded 150 mm Hg. After laminectomy and durotomy at the level of C2, the tumor was resected and the operation completed uneventfully. However, emergence from anesthesia was delayed. On regaining consciousness, the patient was only able to nod her head in response to verbal commands and right-sided hemiplegia was noted. We considered that manipulation of the tumor might be responsible for right-sided hemiplegia. When the tidal volume exceeded 8 mL/kg and BIS exceeded 85, the patient was tracheally extubated and transferred to the recovery room. Because her level of consciousness did not improve, brain computed tomography (CT) was performed 12 h postoperatively, revealing hemorrhage in the vermis and right cerebellar hemisphere (Figure 1). The patient was treated conservatively and the neurological deficits including drowsiness and paralysis gradually recovered. One month postoperatively, the hematoma had been completely absorbed and hemiplegia had resolved leaving a mild residual gait ataxia.

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Figure 1. Plain computed tomographic image of the head 12 h after surgery revealing hematoma in the cerebellar vermis and right cerebellar hemisphere.

Discussion

The most common causes of perioperative intracranial hemorrhage are diverse: use of anticoagulants, high arterial blood pressure, and underlying intracranial vascular abnormalities. However, several case reports have demonstrated that loss of CSF might be another important factor in the pathogenesis of intracranial hemorrhage during spine surgery (1–4). Friedman et al. (4) reported two cases of cerebellar hemorrhage after spinal surgery. They speculated that caudal cerebellar displacement or sag resulting from intraoperative loss of CSF causes transient stretching and occlusion of superior cerebellar veins draining in the cephalad direction into the deep venous system (4).

In the present case, cerebellar hemorrhage was considered to result from loss of CSF after durotomy for the following reasons. First, although anticoagulant therapy is a cornerstone of the treatment of chronic atrial fibrillation, it was not used perioperatively in the present patient. Second, arterial blood pressure was continuously monitored throughout the anesthetic and no episodes of high arterial blood pressure occurred. Relatively mild characteristics of hematoma formation also suggested that less severe stress was responsible for the pathomechanism. Third, magnetic resonance angiography performed postoperatively also failed to demonstrate a vascular lesion.

Because asymptomatic cerebellar hemorrhages are unlikely to be detected and because postoperative cranial CT is not routinely performed after spinal surgery, the incidence of cerebellar hemorrhage may exceed that reported (4). In this case, although tidal volume exceeding 8 mL/kg and BIS more than 85 are controversial criteria for tracheal extubation, we considered the delayed emergence from anesthesia to be a transitory residual effect of fentanyl and propofol. In retrospect, brain CT should have been examined before tracheal extubation.

Cerebellar hemorrhage remote from the site of surgery is seen much more often after supratentorial intracranial surgery, with a reported incidence of 0.08%– 0.29% (5–7). The mechanisms of hemorrhage in spinal surgery resemble those in supratentorial surgery, with either transtentorial pressure gradient as a result of excessive CSF loss or removal of a mass lesion without substantial CSF loss postulated to be responsible for disruption of cerebellar venous blood flow and consequent venous hemorrhage (5,8). Although perioperative factors, such as intraoperative positioning, inherent or induced coagulopathies, and arterial blood pressure, fail to predict the development of remote cerebellar hemorrhage (5), "head-down" positioning during opening of the dura might reduce the risk of this complication, at least in case of spinal surgery (4). In conclusion, it is important to be aware of this complication as a potential causative factor of unexplained neurological deterioration or disturbance on emergence from anesthesia after spinal surgery. Early diagnosis and treatment of this complication are of great importance to patient outcome.

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