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Accidental total subarachnoid block following local scalp anesthesia

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Introduction

Stereotactic biopsy is presently performed routinely in the histological diagnosis of brain tumors before the start of chemotherapy.

We present a case of accidental total subarachnoid block (TSB) [1] in which neurosurgical stereotactic operations were repeatedly conducted under local anesthesia. There are reports of accidental TSB following durapuncture in spinal epidural anesthesia or intentional TSB for the treatment of intractable pain [2]. Our report describes a case of unexpected, temporary coma and apnea that are presumed to have resulted from accidental direct penetration of a local anesthetic agent into the cerebral ventricular system during local scalp anesthesia.

Case report

A 68-year-old woman (weight, 50 kg; height, 142 cm) with the complaint of progressive hemiparesis on the right side was found to have multiple tumors in the brain, and stereotactic biopsy of one of the brain tumors under the guidance of magnetic resonance imaging (MRI) was planned. After subcutaneous infiltration of the scalp using a local anesthetic, the skin was incised and a burr hole (approximately 12 mm in diameter) was made in the left parietal region. The dura was exposed and cut in arcuate fashion. Subsequently, a Sedan side-cutting biopsy needle with a diameter of 2.5 mm was carefully introduced into the brain to the precalculated depth. Several samples were obtained by rotating the outer cannula and slowly withdrawing the needle. Oxidized cellulose (Oxycel, Sankyo, Tokyo) was placed on the dura, and the wound was closed with silk and nylon sutures. The first stereotactic procedure was thus carried out uneventfully, however, postoperative computed tomography (CT) revealed a gradual increase in the size of the hematoma in the left corona radiata, which corresponded to the area targeted for tissue sampling and penetration into the left ventricle. Therefore, an emergency drainage of the hematoma cavity was carried out on the same day. The preoperative setting and targeting for the second stereotactic procedure were the same as those used in the first. A needle was introduced in the same manner and advanced toward the hematoma, and then a drainage tube was advanced along the needle tract and was fixed with sutures on the scalp.

Unfortunately, the drainage tube was accidentally withdrawn on the following day. We were obliged to again introduce a drainage tube into the hematoma cavity with the aid of the stereotactic apparatus. The neurosurgeon administered 20 ml of 1% lidocaine subcutaneously along the previous suture line on the scalp at 15:45. Soon after the infiltration, the patient shouted briefly, became unresponsive to our verbal commands, and stopped breathing. The drapes and stereotactic frame were removed immediately and ventilation with a mask was begun. The blood pressure and pulse were 120/70 mmHg and 65 bpm, respectively; at 15:50, they had increased to 190/120 mmHg and 150 bpm, respectively. Arterial oxygen saturation decreased from 99% to 96%. The trachea was intubated at 16:00. The patient seemed completely paralyzed and did not show any response to intubation. The pupils were fully dilated. The neurosurgeons suspected an additional cerebral hemorrhage and started intravenous administration of mannitol (60 g) and hydrocortisone (300 mg). As sys-

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tolic blood pressure decreased to less than 80 mmHg, dopamine was intravenously infused at the rate of $5\,mg \cdot kg^{-1} \cdot min^{-1}$. Spontaneous breathing, the corneal reflex and the oculocephalic phenomenon were all lost. The level of consciousness was graded as 3 according to the Glasgow Coma Scale. As systolic blood pressure became stable and was kept around 135 mmHg with the infusion of dopamine ($4\,mg \cdot kg^{-1} \cdot min^{-1}$) at 16:10, she was sent for CT examination under controlled ventilation. There was no CT evidence of any new hematoma. She began to show signs of recovering consciousness at 16:40, after she was returned to the ward. The left pupil showed constriction and the left corneal reflex, spontaneous breathing, and responses to verbal commands were restored. She was almost alert at 17:40. Her behavior at 18:00 was almost the same as that before the event. The plasma concentration of lidocaine sampled at 19:00 was $1.47\,\mu g \cdot ml^{-1}$. The hematoma became smaller day by day without drainage. Dilatation of the ventricule did not appear. She received combination therapy with radiation and anticancer drugs under the diagnosis of malignant lymphoma in the brain.

Discussion

The patient in the present case suddenly became comatose during infiltration of the scalp with local anesthetic for the third stereotactic operation. She had orally taken plaunotol (240 mg), famotidine (20 mg) and betamethasone (6 mg) daily. She did not have any history of epileptiform events. The clinical features, such as unresponsiveness, loss of light reflex, pupil dilatation, respiratory arrest, and skeletal muscle flaccidity were temporary, and she gradually recovered from the comatose state within about 1 h. This reversible deep coma resembles the total spinal anesthesia induced by the accidental or intentional injection of a considerable amount of local anesthetic into the spinal subarachnoid space [2]. In total spinal anesthesia, spontaneous respiration ceases, consciousness disappears, and all the reflexes mediated via the cranial and spinal nerves are lost soon after injection of the local anesthetic into the spinal subarachnoid space, while the blood pressure and heart rate remain comparatively stable [2,3]. In general, this comatose state lasts for 1–2 h [2,4], and ventilation must be controlled or assisted during this period. Since a local anesthetic injected subarachnoidally affects the brainstem surface and cranial nerves in addition to the spinal nerves [3], this event is also known as "brainstem anesthesia" [4] or TSB [1]. We have used the term TSB in this report because we consider it to be the most suitable term to describe the event.

In the present TSB, we consider that it is most likely that some of the local anesthetic applied for local scalp anesthesia entered the ventricular system through the existing burr hole and intracerebral path. The small amount of local anesthetic reaching the ventricle presumably then spread out into the subarachnoid space around the brainstem and the upper spinal cord, perhaps affecting not only the cranial nerves and the brainstem surface, but the cervical spinal nerves and spinal cord surface. Tatum and Defalque reported a similar case accompanied with retrobulbar block in which some local anesthetic was thought to be accidentally injected into the brainstem subarachnoid space through the orbita [5].

Bremer, in his classic study with cats, demonstrated that complete deafferentation at the level of the midbrain, i.e., an interruption of all afferent inputs from the spinal and cranial nerves, induced a comatose state [6]. French et al. proposed that an anesthetic state is caused by selective depression of the nonspecific reticular activating system in the brainstem [7]. The clinical signs in our patient suggest that the local anesthetic effect extended to the cranial nerves and even to the upper cervical nerves, but the depth of penetration of the local anesthetic into the surface of the brain-stem and spinal cord is uncertain. Our recent study of spinal evoked potentials revealed that the evoked potentials reflecting the conduction functions of the lateral and dorsal columns disappeared with the initiation of routine lumbar spinal anesthesia (unpublished data). In the present patient, direct depression of the ascending brainstem reticular activating system and the spinal conduction pathways of afferent impulses seem to have contributed to the TSB, in addition to the blockade of the cranial and spinal nerves.

Another possible explanation for the present episode is central nervous system (CNS) toxicity induced by an overdose of the local anesthetic. However, the amount of lidocaine used for local scalp anesthesia in our patient (200 mg) would rarely cause CNS intoxication, even if all of the lidocaine were injected intravenously. However, the plasma concentration of lidocaine at 3 h after the subcutaneous infiltration was high ($1.47\,\mu g \cdot ml^{-1}$). Matsumoto et al. reported that lidocaine injected into the cervical subarachnoid space readily enters into the intracranial subarachnoid space, but that the absorption of lidocaine into the systemic circulation is slow [8]. Burm et al. indicated that local anesthetics in the subarachnoid space are absorbed into the systemic circulation without degradation [9]. Therefore, the high plasma concentration of lidocaine at 3 h after the episode is not inconsistent with accidental TSB. Furthermore, the clinical features observed in this patient were obviously different from those of local anesthetic–induced CNS toxicity. The sympathomimetic symptoms such as transient hypertension and tachycardia observed at the beginning of the episode were presumably

caused by autonomic imbalance between the sympathetic and parasympathetic tone, physical or chemical stimulation by the local anesthetic, and sudden respiratory depression.

In conclusion, we report a rare case of TSB following scalp infiltration of a local anesthetic, some of which is presumed to have entered the subarachnoid space including the ventricular system through an existing burr hole and contiguous intracerebral route made for previous stereotactic procedures. This case called to our attention the possibility that local anesthetic can enter the cerebral subarachnoid space when a neurosurgical procedure is performed under local anesthesia in a patient with a bone defect.

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