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CLINICAL REPORT

Suspected migration of cervical epidural catheter into the brainstem after a difficult catheter insertion

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Abstract We report a case of diplopia during continuous epidural injection presumably caused by catheter migration. A 61-year-old woman underwent shoulder surgery under general anesthesia with cervical epidural anesthesia. The epidural catheter was placed in the C6–C7 epidural space with some difficulty before general anesthesia. The depth of the catheter placed under the skin was 10 cm. On POD 2, the patient noticed diplopia and developed dysarthria despite of good pain control so far. She complained of sudden headache after the rate of continuous epidural infusion was increased to relieve postoperative pain. Computed tomography and T_2-weighted cerebral magnetic resonance imaging revealed an air image and surrounding edema in the pons. Diplopia and dysarthria disappeared after ceasing continuous epidural injection. A 15-cm-long mark under the skin and leak of colorless clear fluid from the puncture site were noted at removal of the catheter. On POD 13, diplopia recurred, which improved gradually. On the 9-month radiologic follow-up, we considered that the symptoms on POD 2 were caused by migration of the epidural catheter into the pons and that her later diplopia was induced by intracranial hypotension syndrome. One should be aware that such an unexpected migration of the catheter can occur following a difficult insertion.

Keywords Diplopia · Migration · Cervical epidural catheter · Intracranial hypotension syndrome

Introduction

Migration of an epidural catheter is rare but is one of the critical complications associated with epidural anesthesia [1, 2]. We report a case of suspected migration of the catheter into the pons followed by diplopia.

Case report

A 61-year-old woman (54 kg, 156 cm) underwent elective arthroscopy of the right shoulder and adhesiolysis under general anesthesia with cervical epidural anesthesia for pain control. Preoperatively she had no abnormality except hypertension and hyperlipidemia. In the sitting flexed position, the procedure was started in the C6–C7 epidural space by the midline approach using a 17-gauge epidural needle (Hakko, Tokyo, Japan) with the bevel directed cephalad. The epidural space was determined at 5.5 cm of needle marker using the loss-of-resistance technique with normal saline without air. The patient complained of shooting pain in the left shoulder and upper arm on catheter insertion. The needle and catheter were simultaneously withdrawn. On the second attempt, she complained of similar pain on catheter insertion. The needle bevel was turned to the right by 30° to advance the catheter. However, she complained of mild pain in the left upper arm; to relieve the dural irritation, we returned the bevel to the left

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by 15° and injected 2 ml 0.375 % ropivacaine through the needle. A radiolucent catheter was then passed 4.5 cm beyond the needle tip without any pain, and the needle was withdrawn over the catheter. None of the fluid was aspirated from the catheter immediately after the insertion of the epidural catheter. There was no respiratory or cardiovascular suppression or neurological abnormality in her upper limbs during the procedure.

General anesthesia was induced intravenously with 100 mg propofol, 100 μg fentanyl, 50 mg ketamine, and 2.5 mg droperidol in the supine position. A laryngeal mask airway was inserted, and anesthesia was maintained with 1.0–1.5 % sevoflurane and continuous epidural anesthesia. In the left lateral decubitus position, 9 ml 0.2 % ropivacaine and 50 μg fentanyl were injected continuously via the epidural catheter at the rate of 2 ml/h with a syringe pump. Her vital signs remained stable, except that systolic blood pressure decreased to 70 mmHg about 30 min after epidural injection started. The rate of the continuous epidural injection was decreased to 1 ml/h to normalize the blood pressure. At the end of the surgery, continuous epidural injection was switched to a disposable infusion pump (COOPDECH Syrinjector; Daiken Medical, Osaka, Japan) containing 97.5 ml 0.1 % ropivacaine and 250 μg fentanyl at the rate of 2 ml/h for postoperative analgesia. The surgery lasted 1 h and 20 min. The patient's vital signs remained stable, and she did not complain of intolerable pain and paralysis in her right shoulder until postoperative day (POD) 2, except for tingles in the left C6 area occurring on POD 1, but epidural solution was changed to 0.2 % ropivacaine only 24 h after surgery (POD 1) because of persistent nausea.

On POD 2, the patient noticed diplopia and dysarthria but did not complain in the morning. The rate of ropivacaine infusion was increased to 4 ml/h because of aggravated pain in the right shoulder at noon. Twenty minutes later, she suddenly complained of headache, dyspnea, and chest discomfort. High systolic blood pressure, up to 190 mmHg, was noted. The rate of continuous epidural infusion was decreased to 2 ml/h immediately, and 10 mg nifedipine was administered orally. The possibility of ischemic heart disease was ruled out because there was no abnormality in her blood tests and electrocardiogram. Then, 2.5 h later, her diplopia and dysarthria worsened, and she informed us about these symptoms for the first time. Continuous epidural injection was stopped. Computed tomography (CT) revealed a very low density

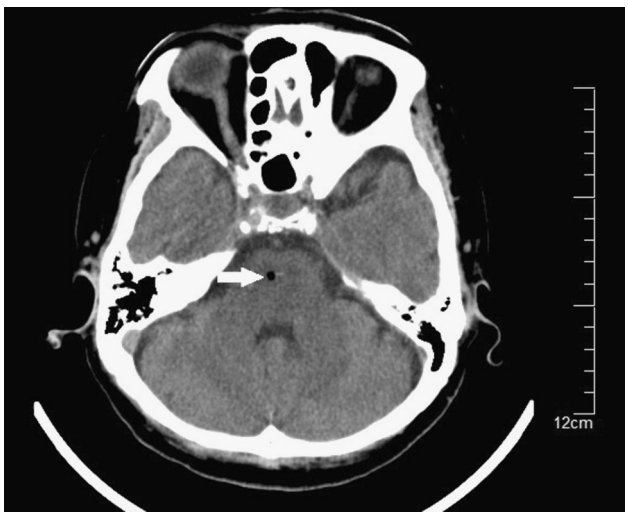

Fig. 1 Computed tomography (CT) image on postoperative day (POD) 2 shows an air "bubble" in the pons (*white arrow*)

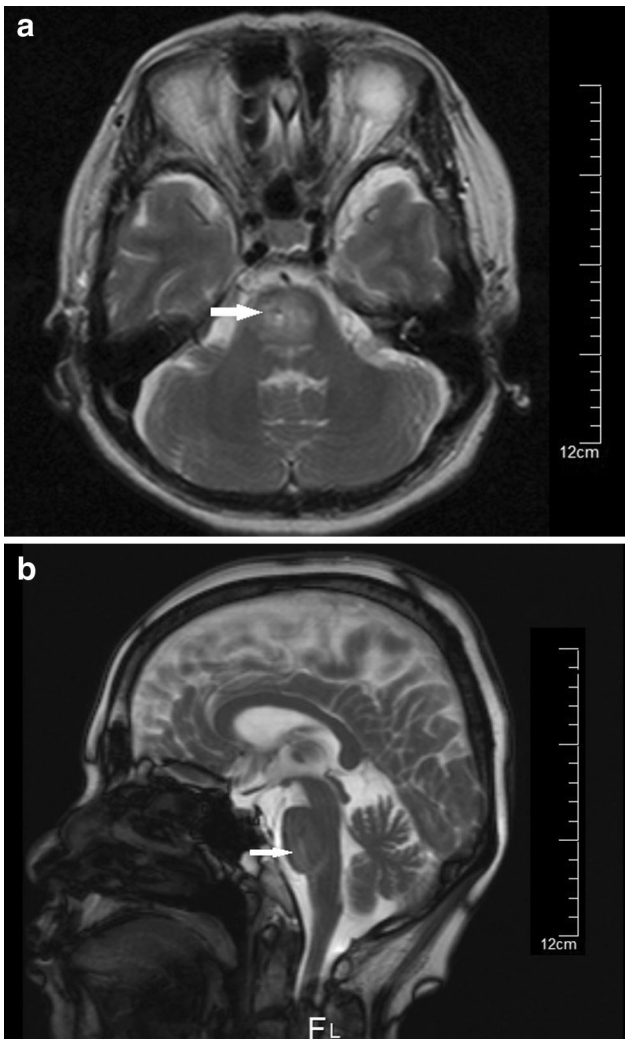

Fig. 2 **a** A T_2-weighted cerebral magnetic resonance image (MRI) on POD 2 shows an air "bubble" and surrounding edema in the pons (*white arrow*). **b** Sagittal view T_2-weighted MRI image on POD 2 shows slight high-intensity area similar to the MRI image performed 9 months after the surgery at the right ventral side of the pons (*white arrow*). This finding was noticed long afterward and was also considered to be caused by an injury resulting from migration of the epidural catheter to the pons

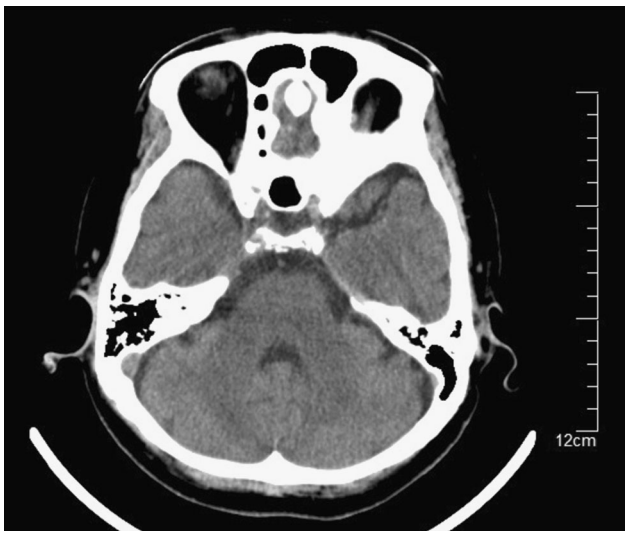

Fig. 3 CT image on POD 13 shows no abnormality

spot in the pons (Fig. 1). T_2-weighted cerebral magnetic resonance imaging (MRI) also showed the low-intensity spot with a surrounding high-intensity area in the pons (Fig. 2a). Diplopia and dysarthria disappeared approximately 1 h after the continuous epidural injection was stopped. When the catheter was removed after CT and MRI, a 15-cm-long mark under the skin and a leak of colorless clear fluid from the puncture site were noted. After removal of the catheter, the patient's vital signs remained stable. We consulted a neurosurgeon and a radiologist about the cerebral lesion on CT and MRI. They could not determine the source of the entry of air and the cause of edema. The patient complained of persistent headache only on moving. A CT obtained on POD 6 showed that the air image in the pons had disappeared.

On POD 13, diplopia progressively relapsed. No abnormality was detected on CT, but only a localized high-intensity spot in the pons was observed on T_2-weighted MRI (Figs. 3, 4). CT and MRI revealed that the air image had disappeared and its surrounding edema had reduced. On POD 31, the patient was discharged. The diplopia improved within 3 months after discharge.

Nine months after the surgery, a high-intensity area was detected at the right ventral side of the pons on T_2-weighted MRI performed during a periodic follow-up examination (Fig. 5). After consulting the neurosurgeon, we identified that the lesion might have been caused by migration of the catheter to the pons.

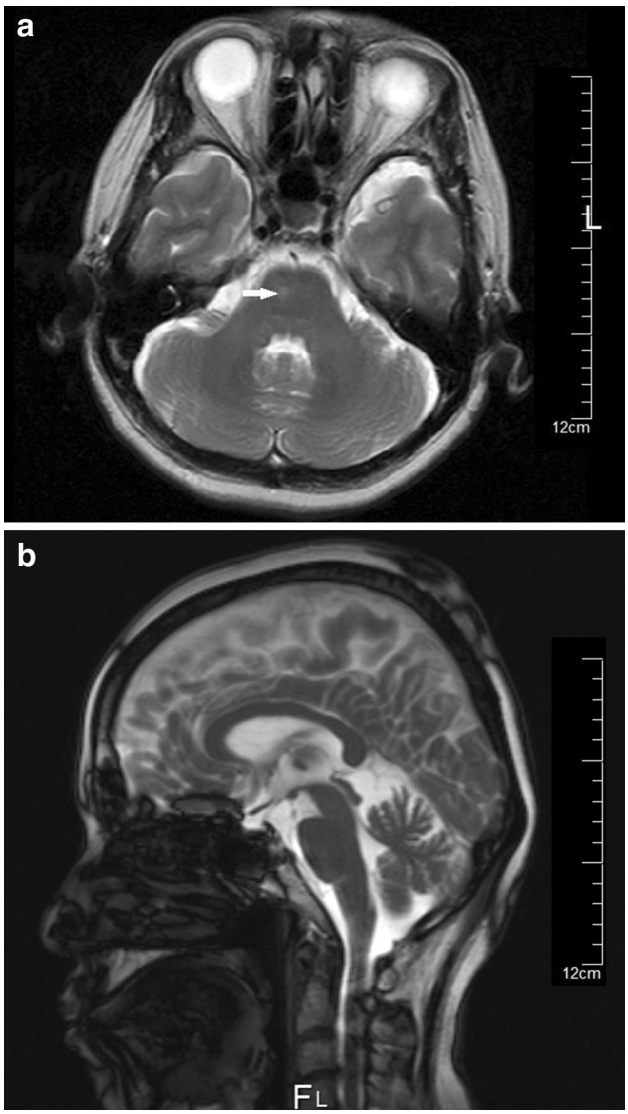

Fig. 4 **a** A T_2-weighted MRI image on POD 13 shows a localized high-intensity spot in the pons (*white arrow*). **b** MRI image on POD 13

Discussion

We encountered a case of diplopia during continuous epidural injection presumably caused by the catheter migration to the pons. Although the course of the catheter was not revealed in an X-ray with contrast medium before removing the catheter, we believe that there are no views explaining a series of symptoms and the following extraordinary radiologic findings except for the catheter migration into the pons. We speculated that diplopia on POD 2 was affected by the catheter migration to the pons, whereas diplopia on POD 13 was caused by intracranial hypotension syndrome (IHS).

As the neurosurgeon suggested, migration of the catheter into the pons and injected local anesthetics might cause diplopia and dysarthria on POD 2 because they disappeared once the continuous injection was stopped. This migration was possibly responsible for the low-density area caused by the air contained in the catheter lumen

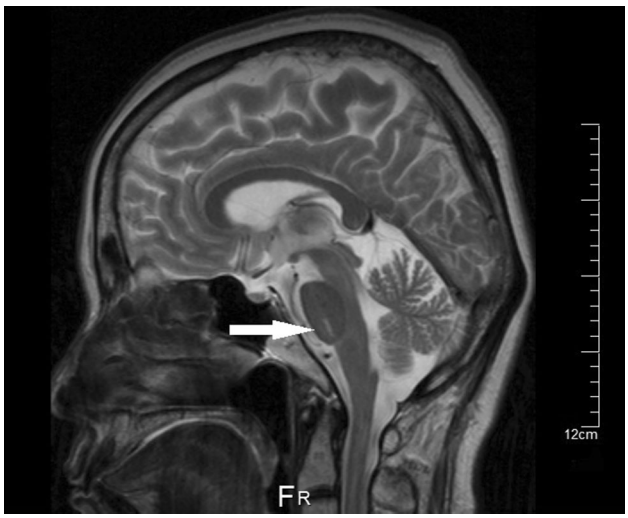

Fig. 5 Sagittal view T_2-weighted MRI image 9 months after the surgery shows a high-intensity area at the right ventral side of the pons (*white arrow*). This finding was considered to be caused by nothing physiological but rather by an injury from migration of the epidural catheter to the pons

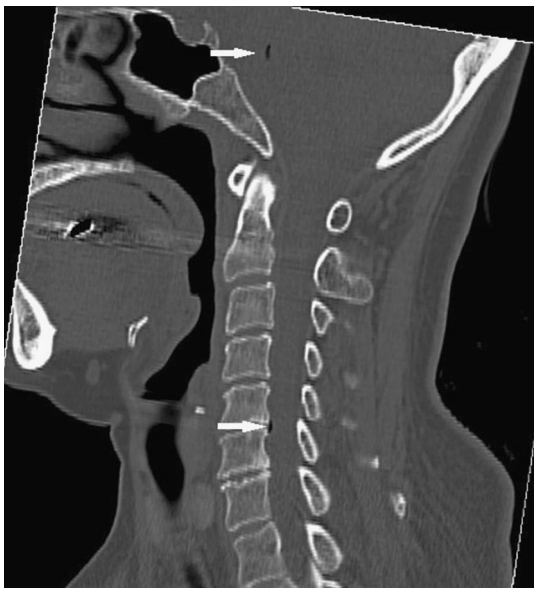

Fig. 6 Sagittal CT image on POD 2 shows two very low density spots (*white arrows*). One (the *upper arrow*) was considered to be located in the pons and it was approximately consistent with a very low density spot in Fig. 1. Another spot (the *lower arrow*) was located in the epidural or subdural space behind the C5 and C6 vertebral bodies. We considered both these lesions were the air either from or inside the radiolucent catheter. This image was also noticed long afterward

or emitted outside (Fig. 1) and the surrounding edema (high intensity) caused by injected local anesthetics (Fig. 2a) on POD 2. We noted that the CT sagittal image on POD 2 (Fig. 6) showed two spots of low density, consistent with air. One area was considered to be in the pons (Fig. 1), and another was in the epidural or subdural space, not in the spinal cord behind the C5 and C6 vertebral bodies. We considered both these lesions were the air either from or inside the radiolucent catheter and the catheter might advance forward and cephalad. Also, we found a slight high-intensity area similar to the MRI image performed 9 months later at the right ventral side of the pons in the sagittal view T_2-weighted MRI image on POD 2 (Fig. 2b). This finding is regarded as nothing intrinsic but is believed to be an injury caused by migration of the epidural catheter to the pons.

Mizoguchi et al. [3] reported a case of epidural catheter migration to the subdural space revealed only by diplopia. In our case, the catheter side ports might be in the epidural space, presenting good pain control, although its blind tip might have compressed the left C6 root on POD 1. Considering the information from that report [3], the catheter might have passed through the dura, advanced forward in the subdural space, and ascended ventrally into the upper subarachnoid space and finally into the pons before the patient's diplopia and dysarthria occurred on POD 2. In addition, dysarthria could be caused by influence on the facial nerve, originating close to the abducens nerve in the pons.

No reports on epidural catheter migration to the brain have been reported. Kao et al. [4] reported permanent paraplegia caused by accidental intracord catheterization for thoracic epidural anesthesia. Generally, catheter migration into the brain is considered to cause severe disabilities. We assume that our case was fortunately less critical because the lesion in the pons might be in a less vascular area.

Angle et al. [1] showed that subarachnoid migration of catheter is unlikely with an intact dura, but unintentional subarachnoid catheter passage is observed if the dura is damaged by the epidural needle. In this case, we slightly turned the epidural needle, which could have damaged the dura, to change the advancing direction of the catheter.

Changing position might be considered to make catheter move 0.5–4 cm inward [3, 5, 6]. We speculated that the catheter might move inward with cervical movements on POD 1 and 2.

We secured the epidural catheter looped at the puncture site using a transparent adhesive dressing (Million Aid Filfix; Kyowa, Osaka, Japan). Because this transparent dressing does not ensure reducing the incidence of catheter migration [3, 6, 7], we should have carefully checked the depth of catheter insertion and the anesthetic area when the patient had any clinical changes.

Diplopia, which relapsed on POD 13 as a result of IHS, was caused by stretching or compression of the abducens nerve secondary to intracranial hypotension as a result of CSF leakage from the dural cleavage after the removal of

the catheter. An ophthalmologist diagnosed her delayed diplopia on POD 13 as abducens paralysis on POD 21. Nishio et al. [8] reported the incidence of diplopia after dural puncture. The period required for the manifestation of diplopia is 1 day to 3 weeks after dural puncture. In our case, diplopia progressively relapsed on POD 13 (11 days after the removal of the catheter) and is compatible with the data presented by Nishio et al.

One should be aware of the possibility of catheter migration into the brainstem with cranial nerve symptoms and development of IHS-related complications such as diplopia. Careful manipulation is required when insertion of the epidural catheter is difficult.

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