Postoperative Transtentorial Herniation of Occult Meningioma after Uneventful Epidural Anesthesia

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Introduction

Reports of transtentorial herniation after neuraxial anesthetic techniques are rare. We report a patient with an occult meningioma who had delayed development of a transtentorial brainstem herniation after uneventful epidural anesthesia for radical prostatectomy.

Case Description

A 55-yr-old ASA I patient was scheduled for radical prostatectomy for prostate cancer under epidural anesthesia. His medical history was tamsulosin. Preoperative review of systems was unremarkable, including the central nervous system. On physical examination, the patient was 178 cm tall and weighed 85 kg. Blood pressure was 130/70 and pulse was 78 beats·min⁻¹. The patient was premedicated intravenously with midazolam 1 mg and fentanyl 50 μg. In the operating room, with the patient in a sitting position, a midline L2-L3 epidural catheter was placed using aseptic technique, without complications by a senior resident on the first attempt. An epidural test dose of 3 ml of 1.5% lidocaine with epinephrine 1:200,000 was negative. Divided doses of 18 ml of 2% lidocaine established an adequate level of surgical anesthesia at the T4-T5 interspace. Epidural blockade was maintained with 0.5% bupivacaine (18 ml over 3.5 hours). The patient was positioned supine with his back extended and his head down to facilitate the surgical exposure. Standard ASA monitors were used throughout the procedures and vital signs documented at 5-min intervals. Over the next 4.5 hours he received intravenously a total of 7 mg midazolam and 300 μg fentanyl for intraoperative sedation. An hour into the procedure, 4 mg of preservative-free morphine sulphate was injected into the epidural space for postoperative pain management. The patient received 4,500 ml of crystalloid and 1 unit of autologous blood intraoperatively with an estimated blood loss of 1,800 ml. Throughout the entire procedure the vital signs were stable and the patient was comfortable. Immediately after surgery, the epidural catheter was removed without complication.

In the post-anesthesia care unit, the patient was awake, alert and fully oriented. Two hours later he was transferred to the inpatient ward with normal mental status, full resolution of his motor block and no clinical signs of hypercapnia. Early in the morning of the day after surgery he was alert and fully oriented, had normal sensation in both lower extremities and was ambulatory. At 1pm the pain team found him somnolent, but arousable, with reactive pupils and able to move all extremities. His respiratory rate was 20 breaths·min⁻¹ and oxygen saturation was 99% on room air. Intravenously naloxone (160 μg) and flumazenil (1.0 mg) in divided doses at 2pm led to no improvement. Arterial blood gases obtained on room air at 3:30pm showed a pH of 7.42, partial pressures of carbon dioxide at 36 mmHg and of oxygen at 108 mmHg, bicarbonate level of 23 meq/L and a base excess of -3. Electrocardiogram, chest x-ray film and electrolyte levels were within normal limits. During the entire time, his vital signs were stable, his extremities were warm and well-perfused and he responded to painful stimuli. At 6pm the patient became stuporous and unresponsive to voice and painful stimuli. The neurologic examination at 7pm revealed decreased movements of all extremities, sluggishly reactive pupils with right pupil slightly larger then left and unremarkable disks. Blood pressure was 159/83 mmHg, heart rate 64 breaths·min⁻¹, respiratory rate 16 breaths·min⁻¹ and oxygen saturation 100% on 3 L·min⁻¹ via nasal cannula. The patient developed Cheyne-Stokes respiration an hour later. His right pupil became fixed and dilated and he was given intravenous mannitol (1g/kg). Upon arrival at the scanner for emergent computed tomography (CT) of his head at 9pm, both his pupils were fixed and dilated and he was unresponsive with a Glasgow coma score of 3. His blood pressure increased to 240/120 mmHg. Simultaneously, his heart rate declined from 120 beats·min⁻¹ sinus tachycardia to 40 beats·min⁻¹ sinus bradycardia, which responded to intravenous atropine. His respiratory rate was 12 breaths·min⁻¹ with an oxygen saturation of 100% and fractional inspired oxygen of 1.0 on AmbuBag. Emergency endotracheal intubation was performed and he was hyperventilated and given intravenous dexamethasone (10mg). The head CT scan showed a 7-cm × 5.5-cm right fronto-temporal mass with central necrosis (Figure 1). Significant mass effect was noted with 2.5-cm midline shift and obliteration of the right lateral ventricle. The patient was transferred to the intensive care unit and an external ventricular drain was placed emergently. The opening intracranial pressure was 28 mmHg. Mannitol and dexamethasone were continued to diminish intracranial pressure. A few hours later the patient was more responsive, able to follow instructions and moved all extremities spontaneously. His pupils were symmetric at 3 mm and reactive. The patient’s family revealed that for several months the patient had complained of headaches and had developed personality changes. Two days after his neurologic decline (three days after prostatectomy) the patient underwent a craniotomy and resection of the mass, which was a benign meningioma. His postoperative course was uneventful. He fully recovered with no residual neurological deficit.

Discussion

We report a case of life-threatening neurological deterioration one day after radical prostatectomy and uneventful epidural anesthesia.
anesthesia due to transtentorial herniation from an occult giant meningioma secondary to an unrecognized dural tear. Etiology of a delayed brainstem herniation may be multifactorial. Intracranial pressure may increase in the immediate postoperative period because of prolonged Trendelenberg positioning, administration of large amounts of intravenous fluids, or hypercapnia secondary to use of sedation and opioids. However, our patient’s hydration status was adequate as assessed by vital signs, examination and urine output. The partial pressures of oxygen and carbon dioxide were within normal limits. The absence of respiratory acidosis suggests that the central effects of epidural morphine administered were not a contributing factor. Our patient did not receive sedatives or opioids after surgery and had no evidence of an acute cardiac or pulmonary event or of metabolic disarrangement.

Although lumbar puncture in patients with space-occupying lesions or increased intracranial pressure is a well-recognized risk factor for development of transtentorial herniation [1-3], reports of transtentorial herniation after neuraxial anesthetic techniques are extremely rare. Eerola et al. [4] reported a case of fatal uncal herniation after spinal anesthesia in a patient with no intracranial process. Su et al. [5] reported a case of fatal brain herniation in a pregnant woman with an occult brain tumor following peripartum epidural anesthesia complicated by unintentional lumbar puncture.

The placement and removal of the epidural catheter in our patient was uneventful. However, the dura may have been punctured during manipulation of the epidural needle or the catheter tip. Our patient received a total of 40 mL into the epidural space. Injection of fluid into the epidural space indirectly increases intracranial pressure [6,7] and may mask a slow cerebrospinal fluid (CSF) leak after dural puncture [5]. This mechanism may explain the delayed presentation of transtentorial herniation in our patient. In the two reports describing herniation after neuraxial anesthetic techniques, the delayed onset of neurological symptoms occurred 11 hours [5] (epidural anesthesia) and 48 hours [4] (spinal anesthesia) after the procedure. The time that elapsed between dural puncture and the onset of mental status changes may reflect the size of the dural puncture and the magnitude of the CSF loss. Although a CSF volume loss can lead to neurological deterioration even in patients without a brain mass [8], patients with space-occupying lesions are clearly at much higher risk for development of transtentorial herniation [1-3].

While this is to the best of our knowledge, the first case of an acute brainstem herniation after uneventful perioperative use of an epidural catheter, this complication might become more common in the future as anesthesiologists care for increasingly aging and complex patients with a higher risk for occult intracranial tumors. For example individuals older than 70 years of age have been found to have a significantly higher incidence of asymptomatic meningiomas [9]. The most common brain tumors, however, are brain metastases, with incidence varying between 15-40%[10], the lung cancer being the most common [10,11]. The preoperative screening for distant metastases in the brain remains however a controversial topic. In a recent report, Shi et al. [12] question the value of a neurological exam in identifying cancer patients with brain metastases, as a high percentage of patients with non–small-cell lung cancer had asymptomatic metastatic spread to the central nervous system. Moreover there were no specific initial T or N staging features of lung cancer on CT that helped identify asymptomatic patients with brain metastases [12].

The relevance of this clinical report is two-fold: First, it wishes to raise awareness that delayed postoperative neurological deterioration in the setting of uneventful perioperative use of epidural anesthesia in patients at risk for occult brain tumors could be related to brainstem herniation. Second, the report suggests that as we practice in an escalating climate of litigation, the anesthesiologist should be aware that at times an extension of the informed consent might be legally prudent to include information about the possible risks of brain herniation for epidural anesthesia in that subset of patients.

Our report does not call for routine preoperative head CT imaging prior to epidural anesthesia and it does not advise to stop the use of epidural anesthesia, a sound and safe technique, in this patient population.

References