



CASE REPORT

What happened? An inexplicable case: Accidental subdural block

Ne oldu? Açıklanamaz bir olgu: İstenmeyen subdural blok

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Summary

Epidural block is an appropriate anesthesia method for outpatient operation. This technique is generally safe and efficient. We present the case of a 20-year-old female patient with accidental subdural block after a single dose of epidural block for pilonidal sinus surgery. Sudden apnea, unconsciousness, and bilateral mydriasis occurred within a few minutes after injection. Within 80 minutes after injection, the patient regained full consciousness and spontaneous respiration, with motor block in lower extremities and sensory block until the T4 level. Motor block continued until 165 minutes after injection, whereas sensory block ended 225 minutes later.

Keywords: Complication; epidural block; subdural block.

Özet

Epidural blok günübürlük operasyonlar için uygun bir anestezi yöntemidir. Bu teknik genel olarak güvenli ve etkili kabul edilir. Bu olgu sunumunda 20 yaşında bayan hastada pilonidal sinüs cerrahisi için tek doz epidural blok sonrası gelişen istenmeyen subdural blok olgusu sunulmuştur. Ani apne, bilinç kaybı ve bilateral midriyazis enjeksiyondan sonra birkaç dakika içinde meydana geldi. Enjeksiyondan sonraki 80.dakikada hastada alt ekstremitelerdeki motor blok ve T4 seviyesindeki duysal blok devam ederken tam bir bilinç açıklığı ve spontan solunumu vardı. Enjeksiyon sonrası motor blok 165 dakika devam ederken duysal blok 225 dakika sonra sona erdi.

Anahtar sözcükler: Epidural blok; komplikasyon; subdural blok.

Introduction

Although epidural anesthesia is a frequently used method for surgical procedures, spinal anesthesia is commonly used for pilonidal sinus surgery because of its more rapid onset. However, spinal anesthesia may be associated with significant side effects, such as hypotension, bradycardia, shivering, nausea, vomiting, significant headache, backache, and urinary retention. The side effects of epidural anesthesia are similar to those of spinal anesthesia but are less common and serious.^[1] A recent study has shown that epidural anesthesia is a suitable anesthetic technique for pilonidal sinus surgery with respect to hemodynamic stability, onset time of analgesia, duration of sensorial block, lack of motor block, side effects, and satisfaction of surgeon and patient.^[2] Subdural block

occurring as a result of local anesthetic spread in the subdural space without dural puncture after epidural block is a rare complication, and its symptoms show similarity with high subarachnoid block.^[3] Diagnosing this complication may be difficult because of diverse symptoms and findings. Insufficient block or high sensory block may be observed following the subdural injection of local anesthetics. Cranial nerves inside cranium may also be affected following subdural injection.^[4]

Herein we present the case of an accidental subdural block, which we had difficulty in identifying following a single dose of epidural anesthesia without leakage of cerebrospinal fluid (CSF) for pilonidal sinus surgery, accompanied by literature.

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Case Report

Written informed consent was obtained from the patient for publication. The patient is a 20-year-old female with the following data: weight, 60 kg; height, 163 cm; ASA I; with no cardiovascular and metabolic disease admitted for pilonidal sinus surgery; erythrocytes, 4.770.000/mm³; hemoglobin, 11.1 g.dL⁻¹; hematocrit, 33.7%; platelets, 184.000/mm³; prothrombin time, 15.4 seconds; PTT, 70.8%; and INR, 1.23. Preoperative other laboratory findings including urine examination, blood sugar, and serum electrolytes were within normal limits. Her general physical examination was normal.

Before entering the operating room, the patient received 500–750 mL of Ringer-lactate solution via intravenous (IV) cannula, and the patient was not pre-medicated. Standard monitoring including non-invasive arterial pressure, electrocardiography, and pulse oximetry (SpO₂) was established in the operating room. Her baseline blood pressure was 110/70 mmHg, pulse was 68/minute, and SpO₂ was 100%. The patient was taken to the sitting position and a single dose of epidural anesthesia was performed in the L3–L4 interspace after cleaning and draping. Epidural space was identified using the loss of resistance technique with an 18G Tuohy needle (Perifix®; B. Braun, Melsungen, Germany). Next, 15 ml anesthetic solution prepared with 10 ml 0.5% bupivacaine and 5 cm³ 2% lidocaine mixture was injected into the epidural space. After the procedure was completed, the patient was taken to supine position again. After few minutes, she had nausea and was unable to move her arms. Considering the possibility of hypotension development, IV 10 mg ephedrine and 0.9% NaCl rapid infusion were immediately injected. At follow-up, blood pressure was 130/75 mmHg and pulse was 145/min. When verbal communication was established with the patient, she told us that she was in a good condition but stressed; therefore, 2 mg dormicum IV (intravenous) was administered as anxiolytic. At this moment, oxygen saturation decreased to 85% and the patient remained in a state of partial unconsciousness. Considering the possibility of a dormicum-related immediate apneic condition and sedation, air was supplied with an O₂ mask. We expected the situation to improve in a few minutes; therefore, we increased the saturation to 100%. After a few minutes, the patient did not respond to

verbal and painful stimuli. She was unconscious and completely immobile; her pupils were dilated. Her arterial blood pressure and heart rate were stable. Oxygenation with a laryngeal mask was provided for 80 minutes. By this time, hemodynamic parameters were normal. At the end of 80 minutes, the patient's consciousness was suddenly regained, mydriatic condition improved and motor movements were regained in hands and arms. Blood pressure and pulse were still stable. When senses were examined with pinprick tests, sensory block was observed until the T4 level and motor block was 3 according to Bromage scale for both lower extremities; in other words, full paralysis was present in the lower extremities. After communicating verbally with the patient for 15 minutes, the patient was taken to prone position and the operation was initiated. The operation continued for approximately 40 minutes, after which the patient was transferred to the recovery room for 45 minutes until motor block disappeared. When motor block disappeared, the patient was transferred to the service unit. After 1 hour, the patient was transferred to the service unit and sensory block had resolved.

Discussion

Anatomically, duramater is composed of elongated and oblate fibroblasts, plenty of extracellular collagens, and cellular junctions. On the other hand, arachnoid layer comprises large cells with many cellular junctions without extracellular space or extracellular collagen. The dural boundary cell layer in the dura-arachnoid junction is characterized by fibroblasts and extracellular space; however, there are slight cellular junctions without extracellular collagen. Due to these formations, when the interior of the dura is compared to its exterior part, a structural weakness is observed. However, under normal conditions, there is no space within dura-arachnoid composition.^[4] The structure, known as dura-arachnoid interface, was defined by Reina et al. using an electron microscope.^[5]

Subdural block development after epidural anesthesia is a rare complication with a space between 0.82% and 7%.^[4] Lubenow et al.^[6] proposed two major (negative aspiration and unexpected extensive sensory block) and three minor (sensory or motor nerve blockade with delayed onset of greater than 10 minutes, a variable motor blockade, and sym-

patholysis out of proportion to the dosage of local anesthetic administered) criteria for the diagnosis of subdural block. They asserted that a subdural blockade should be considered in the presence of both the major criteria and one of the minor criteria. However, there are atypical subdural blockade cases in literature.^[4] In the present case, we initially believed that the present findings may be associated with IV injection or fast absorption of local anesthetics. Bupivacaine IV injection may lead to bradyarrhythmia as well as dysrhythmia, such as hypotension, atrioventricular block, and ventricular fibrillation.^[7] However, absence of attacks, hypotension, bradyarrhythmia, or dysrhythmia in the present case detracted us from this diagnosis. This case met two major criteria for subdural block diagnosis. As the minor criterion, a variable motor blockade was present.

The nerves in the epidural space are surrounded with pia, arachnoid, and dura mater; nerves in the subdural space are surrounded with pia and arachnoid mater; and the nerves in the subarachnoid space are surrounded with only pia mater; therefore, subdural block is a slow onset (approximately 15–20 min) and generally ends after 2 hours.^[4] Subdural space extends from the lower boundary of second sacral vertebra to the base of third ventricle in the cranial cavity, and because it has limited space, apnea and unconsciousness may be observed in a short time after anesthetic solution administration due to intracranial dissemination. The loss of parasympathetic electrical activity arising from Edinger–Westphal nucleus leads to the formation of non-reactive dilated pupils.^[7, 8] In the present case, sudden unconsciousness, apnea, and pupillary dilation were observed as the results of intracranial dissemination of the local anesthetics.

Bradycardia may be caused by cardiac accelerator fibers affected by local anesthetics (T1–T4). However, central volume depletion may have a bigger cardioinhibitory or vasopressor effect. It was thought that block has a minimal effect on sympathetic and motor functions due to the relatively freed of ventral nerve roots. However, hypotension, bradycardia, and more vasodilatation may be observed as a result of parasympathetic activation and/or sympathetic inhibition and accumulation of blood circulation in lower extremities and bowels caused by decreased

venous return.^[4] In the present case, bradycardia and hypotension were not observed. This may be caused by IV ephedrine and fluid administration. Reportedly, ephedrine has positive inotrope and chronotrope effects via β_1 and α_1 adrenergic action.^[9] Additionally, tachycardia developed in our patient.

Being slow and uncommon, the development of motor weakness is associated with progressive respiratory incoordination rather than sudden apnea. The present case shows the result of an unexpected high subdural block with bilateral sympathetic, sensory, and motor blockades. Our patient was unable to move her upper extremities and had apnea. When consciousness was regained after 80 minutes, sensorial block was identified until T4 and the patient was unable to move her feet. Although unilateral block is reported to be common,^[4] our patient had bilateral motor, sensory, and sympathetic blocks. This may be associated with the medication of volume as well as the variation between patients or the extensibility of the space.

The clinic onset and sign of the block in the present case were consistent with high subarachnoid block. However, the absence of CSF via negative aspiration after entering in the epidural space was confirmed; it was shown that subdural block developed with these concomitant findings (loss of consciousness, bilateral pupillary dilation, and sudden apnea), and local anesthetics rapidly dispersed in subdural area toward the cephaloid direction.^[3]

In addition, permanent neurological damage, which is a rare complication, may occur because of the compression of radicular arteries or nerve roots transversely surrounding the epidural space.^[4] Our patient was kept under observation in the postoperative recovery room until block was fully regained, and patient was sent to the service department after it was confirmed that there was no neurological damage. In addition, neurological cases depending on dural injury may be observed after epidural anesthesia (subdural hematoma).^[10] Our findings did not support the diagnosis of subdural hematoma. Because neurological picture completely recovered, performing cranial MR was not deemed necessary in the postoperative period.

Because of diverse clinical findings, it is difficult to reach this diagnosis in advance. The anesthetist should be aware of subdural block, which is a serious complication that may develop during epidural anesthesia and may be life-threatening. It is important to recognize this complication on time and implement appropriate treatments.

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