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CASE REPORT

UNCORRECTED PROOF

What happened? An inexplicable case: accidental subdural block

Ne oldu? Açıklanamaz bir olgu: İstenmeyen subdural blok

Ömer KARACA,[1] Ali AHİSKALİOĞLU,[2] Mehmet AKSOY,[2] Rafi DOĞAN,[1] Elif ORAL AHİSKALİOĞLU[3]

Summary

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

Keywords: Complication; epidural block; subdural block.

Özet

Epidural blok günübirlik 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 duyusal blok 225 dakika sonra sona erdi.

Anahtar sözcükler: Komplikasyon; epidural blok; subdural blok.

Introduction

Epidural anesthesia is a frequently used method for surgical procedures. Due to its more rapid onset, spinal anesthesia is commonly used for pilonidal sinus surgery. 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] In a recent study, it has been shown that epidural anesthesia is a suitable anaesthetic 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 anaesthetic spread in the subdural space without dural puncture after epidural block is one of rare complications and its symptoms show similarity with high "subarachnoid block.[3] Diagnosing this complication may be difficult due to the diversity of symptoms and findings. Insufficient block or high sensory blockage may be observed following the subdural injection of local anaesthetics. Even cranial nerves inside cranium may be affected following subdural injection.[4]

We aimed to present the case of an accidental subdural block which we have difficulty in identifying following a single dose of epidural anesthesia without leakage of CSF (cerebrospinal fluid) for pilonidal sinus surgery and we discussed this case accompanied by literature.

[1]Department of Anesthesiology and Reanimation, Konya Başkent Training and Research Hospital, Konya, Turkey
[2]Department of Anesthesiology and Reanimation, Atatürk University Faculty of Medicine, Erzurum, Turkey
[3]Department of Anesthesiology and Reanimation, Regional Training and Research Hospital, Erzurum, Turkey

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Correspondence ***(İletişim)*****:** Dr. Ali Ahiskalioğlu. Atatürk Üniversitesi Tıp Fakültesi, Anesteziyoloji ve Reanimasyon Anabilim Dalı, 25070 Erzurum, Turkey.
Phone ***(Tel)*****:** +90 - 442 - 344 87 96 **e-mail** ***(e-posta)*****:** aliahiskalioglu@hotmail.com

Case Report

Written informed consent was obtained from the patient for publication. This is a 20-year old female patient, 60 kg, 163 cm, ASA I, with no cardiovascular and metabolic disease admitted for pilonidal sinus surgery; erythrocytes 4.770.000/mm3, haemoglobin 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 (SpO_2) was established in the operating room. Her baseline blood pressure was 110/70 mmHg, pulse was 68/minute, and SpO_2 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. Epi¬dural space was identified by using the loss of resistance technique with an 18G Tuohy needle (Perifix®; B. Braun, Melsungen, Germany). 15 ml anaesthetic solution prepared with 10 ml 0.5% bupivacaine and 5 cc 2% lidocaine mixture was injected into epidural space. After the procedure was completed, the patient was taken to supine position again. A few minutes later, she had nausea and she was unable to move her both arms. Considering the possibility of hypotension development, IV 10 mg ephedrine and 0.9% NaCl rapid infusion were immediately injected. During the follow-up, blood pressure was measured to be 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 and stressful, and 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 O_2 mask. We thought that the situation would improve in a few minutes and; so, the saturation was increased to 100%. In the follow-up of a few minutes, it was observed that the patient did not respond to verbal and painful stimuli. She was unconscious and completely immobile; and her pupils were dilated. The arterial blood pressure and heart rate were stable. Oxygenation with a laringeal mask was provided during 80 minutes. Hemodynamic parameters were normal within this time. 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 stabile. 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 about 40 minutes. After completing the operation, the patient was transferred to the recovery room for 45 minutes until motor block disappeared. When motor block disappeared, the patient was sent to the service unit. After an hour, the patient was visited in the service unit and sensory block was observed to have resolved.

Discussion

Anatomically duramater is composed of elongated and oblate fibroblasts, plenty of extracellular collagens, and cellular junctions. On the other hand, arachnoid layer consists of large cells with many cellular junctions without extracellular space or no 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 called as dura-arachnoid interface was defined by Reina et al., by 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 (a negative aspiration and unexpected extensive sensory block) and three minor (sensory or motor

nerve blockade with delayed onset of greater than 10 minutes and a variable motor blockade, and sympatholysis out of proportion to the dosage of local anaesthetic 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 our case, we first thought that the present findings may be associated with IV injection or fast absorption of local anaesthetics. Bupivacaine IV injection may lead to bradyarrhytmia as well as dysrhytmia such as hypotension, atrioventricular block, and ventricular fibrillation.[7] However, absence of attacks, hypotension, bradyarrhytmia or dysrhytmia in this 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.

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

Bradycardia may be observed due to the cardiac accelerator fibres affected by local anaesthetics (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. But, 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 our case, bradycardia and hypotension were not observed. This may be caused by IV ephedrine and fluid administration. It was known that ephedrine has positive inotrope and chronotrope effects via β1 and α1 adrenergic action.[9] Even, tachycardia developed in our patient.

Being slow and uncommon, the development of motor weakness is associated with progressive respiratory incoordination rather than sudden apnoea. This presentation 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 she had apnoea. When consciousness was regained after eighty 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 our case were consistent with high subaracnoid block. However, both the absence of CSF via negative aspiration after entering in the epidural space was confirmed and it was shown that subdural block developed with these concomitant findings (loss of consciousness, bilateral pupillary dilation, and sudden apnoea) and local anaesthetics rapidly dispersed in subdural area towards the cephaloid direction.[3]

In addition, permanent neurological damage -a rare complication- may occur due to the compression of radicular arteries or nerve roots surrounding epidural space transversely.[4] Our patient was observed 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. Also neurological cases depending on dural injury may be observed after epidural anesthesia (subdural haematoma).[10] The findings that we encountered detracted us from subdural haematoma. Because neurological picture completely recovered, cranial MR was not found necessary in the postoperative period.

Due to the diversity of clinical findings, it is difficult to identify this diagnosis in advance. The anaesthetist 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 recognise this complication on time and implement supportive treatments.

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