Transient aphasia following spinal anaesthesia in an orthopaedic patient

Tripat B, MD, Assistant Professor
Ruchi G, MD, DNB, Professor and Head
Sonika T, MD, Assistant Professor
Department of Anaesthesia and Critical Care, Sri Guru Ram Das Institute of Medical Sciences and Research Amritsar, India
Correspondence to: Bindra Tripat, e-mail: tripat_noori@yahoo.co.in
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Abstract

A 50-year-old male [American Society of Anesthesiologists (ASA) grade II] was scheduled for lower limb orthopaedic surgery. The subarachnoid space was localised with difficulty at L3/4 interspace and 3 ml of hyperbaric bupivacaine was given. Within a few minutes, the patient developed aphasia with a very high sensory block extending to C2 dermatome, followed by apnoea. The patient remained haemodynamically stable throughout surgery and respiration resumed within five minutes. Inadvertent subdural deposition of local anaesthetic was speculated to be the cause of this unusual presentation.

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Introduction

Spinal anaesthesia is commonly used for infraumbilical surgeries. Although it is considered to be a safe and reliable technique, the actual spread of block depends on many factors.[1] Occasionally, during the performance of a neuraxial block, an unexpectedly high or low level of block is achieved due to accidental injection of local anaesthetic into a meningeal plane other than that desired.[2] Even cardiac arrests have occurred with unexpected speed against a background of apparently stable haemodynamics.[3] A greater awareness of the variable presentations is important as timely management is vital to avoid potentially critical complications. We report a case in which, during spinal anaesthesia, the patient a developed aphasia and apnoea with an unexpected high level of sensory block while maintaining remarkably stable haemodynamics.

Case report

A 50-year-old male, who was 167 cm tall and weighed 70 kg, was scheduled for Illizarov fixator for non-union of proximal tibia. He was a known diabetic and had been controlled on oral hypoglycaemic agents for the last five months. There was no history of any other significant medical condition, in particular psychiatric illness or any previous surgery. His general physical examination and all routine investigations were unremarkable.

In the operation theatre, monitors were attached to the patient and his vital signs were recorded. Peripheral venous access was established and a normal saline infusion commenced. Subarachnoid block was attempted unsuccessfully with 26G Quincke needle at the L3/4 and L2/3 interspace in midline in the sitting position. A third attempt at L2/3 interspace with 23G Quincke needle, using a paramedian approach, was successful and 3 ml of hyperbaric bupivacaine was administered once a free flow of cerebrospinal fluid was achieved. The patient was placed in a supine position with a single pillow to support the head and O_2 was given via face mask at 5 l/minute.

Within two minutes, the patient began to feel very weak and lightheaded. His radial pulse was normal in rate, rhythm, as well as volume, with a heart rate (HR) of 96 beats per minute (bpm). His blood pressure (BP) was 138/72 mmHg and oxygen saturation (SpO_2) was 98%. The patient suddenly became aphasic and was unable to move his arms, but communicated by making facial expressions. A rapid examination revealed that the sensory level of the block had risen to C2. Shortly thereafter, he yawned before becoming apnoeic, accompanied by a brisk fall in SpO_2 to 80%. The patient was ventilated with 100% O_2 using bag and mask and SpO_2 increased to 100%. While we were preparing to intubate him, the patient commenced

breathing spontaneously, but his efforts were shallow and his breathing had to be assisted for a further five minutes. Throughout this period, his HR remained around 80 bpm and BP averaged around 130/80 mmHg. Five minutes later, his spontaneous breathing efforts had improved sufficiently to allow the bag and mask to be replaced by a ventimask. Fifteen minutes into the incident, the patient was able to speak. By 30 minutes, sensory level of block had receded to T4. The surgery went on uneventfully for 90 minutes. He was awake, alert and all modalities of sensation had returned by two hours. The incident was not followed by any residual neurological deficit or postspinal headache.

Discussion

Spinal anaesthesia conducted under routine conditions and in a standard manner carries a poorly understood risk of sudden cardiac arrest and severe brain injury in healthy patients.[3] The noteworthy problems encountered in this nonsedated patient, who received a subarachnoid block in the sitting position, were a very high sensory block with transient apnoea, aphasia, and motor weakness of the upper limbs. Despite this, the patient failed to develop the significant hypotension, bradycardia or pupillary dilatation that is normally suggestive of high spinal anaesthesia with associated sympathetic block.[2,4]

Extensive sensory blockade with sparing of sympathetic functions is the most common presentation of inadvertent subdural injection.[2] Motor weakness is usually slow to develop and less profound. Many other presentations of subdural block have been reported in the literature, such as failed spinal anaesthesia, significant motor weakness of the upper extremities and intercostal muscles, Horner's syndrome, delayed or faster than usual onset of block and significant hypotension.[2] This is due to the anatomy of the subdural space. A study of the ultrastructure of the subdural space using electron microscopy has been helpful in explaining the wide inter-patient variability that is seen with subdural blocks. The subdural space extends into the cranial cavity and laterally over the dorsal nerve roots. The dura and arachnoid are attached together on the ventral root, making the space very small ventrally. This causes relative sparing of the ventral nerve roots and pooling of drug in the dorsal segment.[2,4]

Loss of consciousness with remarkable haemodynamic stability has been reported in parturients after spinal anaesthesia for Caesarean section, by Chan et al and Bhati et al. Subdural extension was speculated to be the cause for the unusual presentation. Since the subdural space extends cranially, local anaesthetic block of the brainstem is possible, causing loss of consciousness and apnoea.[5,6] The apparent aphasia in our patient could have been caused by the effect of bupivacaine on the brainstem cranial nerves. Subdural catheterisation leading to trigeminal nerve palsy has also been reported, signifying cephalad spread of the anaesthetic agent into the cranial cavity.[7] Acute mental status change and aphasia after labour analgesia with intrathecal sufentanil/bupivacaine has been reported and explained by the likely cephalad spread of intrathecal sufentanil.[8]

Compression of the thecal sac, spinal canal abnormalities and difficulties with block placement are risk factors for total spinal block or accidental subdural injection. We had difficulty in locating the subarachnoid space as the needle repeatedly encountered bony resistance. It is presumed that a spinal needle may pierce the dura, as well as the arachnoid, so that it lies partly in both the subarachnoid and subdural spaces. Cerebrospinal fluid leaking into the subdural space may distend the space, causing drugs to preferentially distribute in the subdural rather than the subarachnoid space.[5] Long-bevelled needles also increase the chance of partial placement of local anaesthetic in the subdural space.

Most of the cases of inadvertent subdural deposition of local anaesthetics while attempting central neuraxial blocks have been reported in patients undergoing Caesarean section. Although we do not have any radiological confirmation, we speculate that in this case, subdural extension of the local anaesthetic was the most likely cause of the unusual presentation. In cases of extensive sensory blockade, the possibility of a subdural block should always be considered to ensure that its life-threatening complications are managed timeously.

References

1. Cook TM, Counsell D, Wildsmith JAW. Major complications of central neuraxial block: report on the Third National Audit Project of the Royal College of Anaesthetists. Br J Anaesth. 2009;102(2):179-190.
2. Agarwal D, Mohta M, Tyagi A, Sethi AK. Subdural block and the anaesthetist. Anaesth Intensive Car. 2010;38(1):20-26.
3. Caplan RA, Ward RJ, Posner K, Cheney FW. Unexpected cardiac arrest during spinal anaesthesia: a closed claims analysis of predisposing factors. Anesthesiology. 1988;68(1):5-11.
4. Soni W, Holland R. An extensive lumbar epidural block. Anaesth Intensive Care. 1981;9(2):150-153.
5. Stevens RA, Stanton-Hicks MA. Subdural injection of local anaesthetic: a complication of epidural anaesthesia. Anesthesiology. 1985;63(3):323-326.
6. Chan YK, Gopinathan R, Rajendram R. Loss of consciousness following spinal anaesthesia for caesarean section. Br J Anaesth. 2000;85(3):474.
7. De la Gala F, Reyes A, Avellanal M, et al. Trigeminal nerve palsy and Horner's syndrome following epidural analgesia for labour: a subdural block? Int J Obstet Anesth. 2007;16(2):182.
8. Fragneto RY, Fisher A. Mental status change and aphasia after labor analgesia with intrathecal sufentanil/bupivacaine. Anesth Analg. 2000;90(5):1175-1176.