Accidental deposition of local anaesthetic in the subdural space following caudal block

Prachee Sachan, MBBS, Postgraduate; **Veena Asthana** MD, Associate Professor; **Sanjay Agrawal** MD, PDC, Associate Professor
Department of Anaesthesia, Himalayan Institute of Medical Sciences, Dehradun, India
Correspondence to: Sanjay Agrawal, e-mail: drumstix1972@yahoo.co.in
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Abstract

The incidence of accidental injection of local anaesthetic into the subdural space during neuraxial blockade is rare. The presentation of unexplainable clinical signs that do not match the clinical picture of subarachnoid or intravascular injection of the local anaesthetic agent should invoke high suspicion of unintentional subdural block. We report on a case of a six-month-old infant who developed motor block and unconsciousness with haemodynamic stability, following a caudal block for postoperative analgesia. The report will help to illustrate the mechanism behind the complication of subdural deposition of the drug, its detection, treatment and possible avoidance.

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Introduction

Caudal epidural block is a technique that is utilised as a supplement to general anaesthesia for postoperative analgesia, especially in paediatric patients undergoing surgery that involves infraumblical, perineum and lower extremities.

The subdural space is a space between the dura and arachnoid mater, containing a minute quantity of serous fluid.[1] It is a potential site for accidental injection of a local anaesthetic agent during the performance of the neuraxial block. Accidental subdural deposition of the local anaesthetic occurs rarely and few anaesthesiologists are familiar with its clinical presentation.[2-4] We report on a case of suspected subdural deposition of local anaesthetic in an infant who was administered caudal anaesthesia for postoperative pain relief. The case report and a review of literature are presented.

Case report

A six-month-old female infant, weighing 6 kg was brought to the orthopaedic outpatient department of our hospital with a visible deformity of the right lower limb, following trauma three months previously. An X-ray of the lower limb demonstrated malunited fracture of tibia. A surgical procedure of osteoclasis with cast application under anaesthesia was planned. Preoperative evaluation revealed an uneventful birth history, and normal milestones as per the age, and haematological and biochemical investigations appropriate for the age. The infant was kept nil *per os* for four hours prior to surgery. Written informed consent was taken from the parents and the infant was premedicated with phenothiazine syrup 1 mg/kg 30 minutes prior to surgery.

In the operating room, standard monitoring, including an electrocardiogram, noninvasive blood pressure measurement and oxygen saturation (SpO_2), was attached and anaesthetic induction performed with incremental concentrations of sevoflurane in oxygen. Intravenous access was established and an injection of fentanyl 2 µg/kg and that of atracurium 0.5 mg/kg was administered. A laryngeal mask airway (LMA) size 1.5 was placed and anaesthesia maintained with 66% nitrous oxide (N_20) in oxygen (O_2) and sevoflurane. The surgical procedure lasted 45 minutes. As the effect of the atracurium wore off and the infant regained spontaneous respiratory efforts, she was maintained on spontaneous respiration with 66% N_20 in O_2 and sevoflurane 4-5% to administer caudal block for postoperative analgesia in the left lateral position. The caudal block was administered utilising a 24 G hypodermic

needle. The space was confirmed by the "swoosh" test[5] and 4 ml of 0.25% bupivacaine was administered slowly over five minutes.

The infant was turned supine and the LMA removed. She moved her limbs and breathed spontaneously. While preparing for the transfer to the post-anaesthesia care unit (PACU), it was observed that the patient's motor activity gradually diminished, and 10 minutes later, the infant stopped moving her limbs and stopped responding to painful stimuli and became apnoeic. Bag-mask ventilation was started utilising the Jackson Rees circuit. She was intubated with an uncuffed endotracheal tube size 4 (internal diameter) and intermittent positive pressure ventilation continued. The patient was haemodynamically stable with blood pressure (BP) of 90/40 mmHg and a heart rate (HR) in the range of 130-150 beats/minute. As no respiratory effort or spontaneous movements were present after 30 minutes of observation, it was decided to shift the patient to the intensive care unit for elective ventilation. The BP and HR were stable during the period. The infant was put on synchronised intermittent mandatory ventilation (SIMV) with a tidal volume of 50 ml, a respiratory rate of 24/minute and fraction of inspired oxygen 0.5. The infant was pink in colour, maintaining SpO_2 of 100%, with normal-size pupils and reactions. Over the next 60 minutes, she started moving her limbs again and regained spontaneous respiration. The SIMV rate was gradually tapered off and the patient weaned to the continuous positive airway pressure mode of ventilation. On achieving good respiratory effort, movement of her limbs and stable haemodynamics, the infant was extubated. She was kept overnight for observation and transferred to PACU the next day. Her remaining stay in the hospital was uneventful.

Discussion

Epidural block occasionally exhibits an atypical pattern of spread. This may be caused by accidental injection of the local anaesthetic into the subarachnoid or subdural space. An unintended subarachnoid injection following an epidural manifests as profound motor block and unstable haemodynamics, especially bradycardia and severe hypotension,[6,7] with apnoea or respiratory difficulty.

The subdural space is an anatomical potential space extending from the cranium to the lower body of the second sacral vertebra, and extending laterally over the exiting dorsal roots. The arachnoid and dura mater are fixed at different points over the dorsal root ganglia extending into this potential space. By contrast, the meninges are firmly attached over the ventral roots. This causes subdural injections of the local anaesthetic to pool in the posterior segment, with relative sparing of the anterior nerve roots that carry sympathetic and motor fibres.[8,9]

Migration of the local anaesthetic into the subdural, extrathecal space, or the dura-arachnoid interface, allows the movement of the local anaesthetic in the cephalad direction and into the thoraco-cervical segments. Injection under pressure, generated by the force of the injection, permits the movement of the injectate into the path of least resistance, resulting in absorption of the solution across the epithelium. An uneven block, with an unpredictable spread, results in sensory and motor blockade in disproportion to the expected level. Many researchers and clinicians believe that this phenomenon of unintended subdural deposition of the local anaesthetic is a result of microscopic trauma during placement of the epidural catheterisation and extension of the epidural block.[10]

The incidence of subdural deposition of the local anaesthetic during the neuraxial block is 0.82%.[11] Recent studies indicate that the incidence is much higher, ranging from 1-13%. The diagnosis of subdural blocks is difficult on a clinical basis because of their varied presentation.[12]

Lubenow et al described the major and minor criteria for the diagnosis of subdural block.[11] Major criteria included a negative aspiration test and an unexpected extensive sensory block. Minor criteria included delayed onset, variable motor block and sympatholysis out of proportion to the administered dose of the local anaesthetic. The presence of both major criteria and one minor criterion is important for the detection and suspicion of subdural injection of the local anaesthetic.

Hoftman and Ferrante proposed a four-step diagnostic algorithm, based on the clinical presentation of the radiologically proven cases of subdural block (Table I).[13] The first step involves the tactile feel of the performer during accomplishment of the epidural block and the absence of cerobrospinal fluid. The second step includes assessment of the block and its classification as excessive, restricted, or neither. The third step is based on clinical criteria, such as delayed onset of block (> 20 minutes), cardiovascular stability, motor sparing, patchy or asymmetrical spread, respiratory failure and cranial nerve involvement. The fourth step involves confirmation of the subdural placement of the local anaesthetic radiologically, using an X-ray,[14,15] computed tomography scan or magnetic resonance imaging.[16] The presence of injected fluid in the space confirms subdural location. Incorporating the four criteria increases the sensitivity of diagnosing the subdural placement of the local anaesthetic.

A patient with accidental deposition of the local anaesthetic into the subdural space should be monitored closely and patients reassured that this is a reversible and temporary event.

Table I: Criteria for diagnosing the subdural deposition of the local anaesthetic

Lubenow's criteria	Hoftman and Ferrante algorithm
Major	**First step**
Negative aspiration test	Tacticle feel of the performer and the absence of cerebrospinal fluid
Unexpected extensive sensory block	
Minor	**Second step**
Delayed onset of the block (> 10 minutes)	Assessment of the block and classification into "excessive", "restricted" or "neither"
Variable motor block	
Sympatholysis out of proportion to the dose of the local anaesthetic	
The presence of both major criteria and one minor criterion is required for detection and suspicion of subdural block	**Third step**
	Delayed onset of the block (> 20 minutes)
	Cardiovascular stability
	Motor sparing and a patchy spread
	Respiratory failure and cranial nerve involvement
	Fourth step
	Confirmation by X-ray, computed tomography scan or magnetic resonance imaging

Current data support the use of ultrasound guidance while peripheral nerve blocks are being performed. However, use of ultrasound assistance for paediatric neuroaxial blocks is controversial, and not enough supporting evidence is present to issue a general recommendation on its routine use.[17]

In our case, the patient developed sudden loss of movement, unconsciousness, and stable BP and HR. Sensory block could not be assessed as the patient was very young. Early recovery of the infant with full neurological recovery favours subdural deposition of the drug. The slow onset of the block, together with the absence of cerebrospinal fluid in the needle hub during the performance of the block, were major factors against the deposition of the drug in the subarachnoid space. Cardiovascular instability is usually not seen in infants[18] after a subarachnoid block, but may be a diagnostic criterion in adults. Our patient may be the youngest in the literature where probable subdural deposition of local anaesthetic occurred during a caudal epidural block.

Conclusion

Anaesthesiologists should be aware of the possibility of subdural block during central neuraxial anaesthesia. Differential diagnosis should be considered in cases of extensive sensory blockade, despite apparently small volumes of epidurally administered local anaesthetic, unexpected failure of the block, or atypical presentations following an otherwise uncomplicated regional block. Giving assurance to the patient and conducting careful monitoring is useful in such cases.

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