Subdural block and Horner’s syndrome following caudal anesthesia

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Abstract
We report a case of subdural block and Horner’s syndrome after caudal anesthesia in a child with left impalpable testis, who needed second stage orchiopexy. In literature Horner’s syndrome was seen after caudal and total spinal anesthesia but Horner’s syndrome together with subdural block right after caudal anesthesia is very rare. Anesthesiologist should be aware and alert such type of an unexpected complication of caudal anesthesia for uneventful overcome.

Keywords: Caudal anesthesia; subdural block; Horner’s syndrome.

Introduction
Larger doses of local anesthetics associated with caudal or epidural anesthesia may result in symptoms like high spinal block and Horner syndrome (1). Most anesthesiologists do not use caudal anesthesia due to fear of such unexpected complications. We wish to share our experience in a case of subdural block and concomitant Horner’s syndrome.

Case report
A 23-month-old boy was admitted to our center with left impalpable testis requiring second-stage orchiopexy 6 months after the first stage operation. His medical history was otherwise unremarkable. The friendly patient was taken to the operating room without premedication. ASA standard monitoring was placed with initial vital signs BP 86/48, HR 99, O₂ sat 99%. Uneventful inhalational induction was done with oxygen 40%, air 60% mixture and incremental increased sevoflurane. A 24 gauge intravenous cannula was placed in left hand and 1 mcg/kg fentanyl and 0.6 mgr/kg rocuronyum were given to patient for induction. The patient was intubated and anesthesia was maintained with sevoflurane-air-oxygen mixture. During the retroperitoneal exploration, the left atrophic testis was founded and orchiectomy was performed. At the end of the operation while spontaneous ventilation was noticed in ventilator monitor the patient was turned to left lateral fetal position for caudal block. Caudal block performed
without difficulty and 14 cc of 0.25% bupivacaine injected after negative aspiration for blood and cerebrospinal fluid. Vital signs were Blood Pressure (BP) 85/50, Heart Rate (HR) 101, Respiratory Rate (RR) 34, O2 saturation 100% on air/O2 (FIO2 40%) and sevoflurane dial 2%. While turning patient supine after the caudal block was completed; his respiratory rate was 28. Ten minutes later we noticed the patient became apneic an his vital signs now BP 80/49, HR 82. After twenty minutes the patient had hiccup and during this time we noticed bilateral pupillary dilatation thereafter miosis of the left eye. About 60 minutes after the caudal injection we noticed spontan ventilation of the patient and in 90 minutes, the patient was awake. BP 90/53, HR 102, RR 30. Left pupil miosis continued and left eye was ptotic. The patient was extubated and followed up in postanesthesia care unit. Ptosis and miosis of the left eye was explained as component of Horner syndrome. In the 3rd hour after the surgery miosis and ptosis of the left eye were lost.Patient can walk without his mother’s aid.

Discussion

Subdural space is a potential space found between the dura and arachnoid mater. It contains small amount of serous fluid and extends intracranially. The space extends from lower border of the second sacral vertebra into the cranial cavity, unlike epidural space (1). Local anesthetics can travel higher in the subdural space than in epidural space. Larger doses of local anesthetics associated with caudal or epidural anesthesia may result in symptoms like high spinal block and Horner syndrome (2). Horner’s syndrome has been described classically as unilateral ptosis, miosis, anhydrosis. It seems to be benign in most cases but due to high sympathetic block, hemodynamic instability and respiratory compromise must be concerned. Treatment is the same as high neuroaxial blockade such as intubation, mechanical ventilation and cardiovascular support. Since aspiration will generally be negative prevention is difficult. Horner’s syndrome occurs with interruption of the sympathetic nerve fibers to the pupil, levator palpebrae, conjunctiva and face. Most appropriate theory about mechanism of Horner’s syndrome with caudal or epidural anesthesia is cephaled spread of local anesthetics that cause high sympathetic blockade (3,4). We can explain some of neurologic findings as subdural spread of high dose local anesthetics cause apnea and early bilateral pupillary dilatation then with time the level of block will lower so spontaneous ventilation of patient was noticed. But exact mechanism what causes continuation of the unilateral sympathetic block at least the T4 level which could be explained by patient positioning (dependent side) during caudal injection. When a total spinal occurs the nerve supply to the diaphragm (cervical roots C 3-5) is blocked and respiratory failure develops rapidly. But sudden respiratory arrest may also be caused by hypoperfusion of the respiratory centers in the brainstem. A wide variability in the onset time of subdural block, from 7 to 30 minutes, has been attributed to the amount of local anesthetic deposited in the subdural space (5). Delayed onset of apnea as a sign of high block is also strongly suggestive of a subdural block in our patient. We did not see any hypotension and bradycardia after performing caudal anesthesia. This signs may look controversial to high sympathetic block but numerous reports exist of infants tolerating high or total spinal anesthesia without the significant autonomic changes seen in adults. Although the reasons for these finding are not clear this may be the cardiovascular stability in infants is due to either a smaller venous capacitance in lower extremities (less pooling of blood) or relative immaturity of sympathetic nervous system which result in less dependence on sympathetic vasomotor tone (6). In many cases Horner’s syndrome was observed after caudal anesthesia or total spinal anesthesia (7) but our case is particularly rare due to occurrence of subdural block and concomitant Horner’s syndrome. This case scenario can be explained as some part of local anesthetic spread subdurally but remaining epidurally to
higher levels with probably accidental minor trauma of needle to the dura mater. But with careful examination and close monitoring anesthesiologist can overcome this nightmare and will continue this effective neuroaxial analgesic method for postoperative analgesia. The anesthesiologist should also know the complications of this analgesic method to overcome easily and to give information about them to the patients.

References