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Bladder Spasm in Children after Surgery for Urethroplasty: A Case Report

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Abstract

The incidence of bladder spasm in children with hypospadias treated with caudal block before surgery is uncommon. We presented a case of bladder spasm after urethroplasty surgery that was successfully treated with anisodamine without any sequelae. The caudal block plane may only reach the sacrum and does not reach the bladder wall. I suggest a local anesthetic drug dose of 1 ml/kg, totaling not more than 20 ml, to avoid the occurrence of postoperative bladder spasm and increase the comfort of children.

Keywords

Bladder Spasm, Hypospadias, Caudal Block, Case Report

Introduction

Bladder spasm refers to the involuntary contraction of the detrusor bladder muscle [1]. It commonly occurs due to catheter stimulation of the bladder wall and can be challenging to alleviate with opioid analgesic drugs. To address this issue, caudal block and lumbar epidural block are potential interventions for relieving spasms [2,3]. However, cases of bladder spasm following caudal block are uncommon. This paper presents a case of postoperative bladder spasm in a child who underwent caudal block.

Case Presentation

The child was a 5-year-old male, weighing 34.5 kg, and measuring 131 cm in height. Five years ago, he received a diagnosis of hypospadias due to the ventral location of the urethral opening. The patient did not experience frequent urination or painful urination. This time, the plan was for the patient to undergo urethroplasty. In the operating room, standard electrocardiogram and oxygen saturation (SpO_2) monitoring were implemented. Following the conventional induction of anesthesia, the patient was intubated using propofol (70 mg) and midazolam (2 mg) for sedation, sufentanil (7.5 μg) for analgesia, and cisatracurium (3 mg) for muscle relaxation. A caudal canal block was then performed using 15 ml of 0.2% ropivacaine and 5 μg of epinephrine. Throughout the operation, a continuous infusion of 3% sevoflurane was used to maintain anesthesia, and no additional analgesic drugs were administered.

The vital signs remained stable during the operation, which lasted for 1.2 hours. After the surgery, the child regained spontaneous breathing, and atropine and neostigmine were administered. The tracheal catheter was removed once the tidal volume reached 200 ml, $EtCO_2$ was maintained at 40-45

mmHg, and the swallowing reflex had returned. Following extubation, the child experienced mild laryngospasm, which was alleviated through mask pressure ventilation after receiving a 20 mg intravenous infusion of propofol. Approximately 10 minutes later, the child woke up but became extremely agitated and attempted to remove the catheter on their own. After ruling out factors such as hypoxia and hypercapnia, it was determined that the child's restlessness during the recovery period was due to pain.

Subsequently, the child fell asleep peacefully after receiving sedative and analgesic drugs. When the child woke up again, they remained agitated and reported the desire to urinate along with pain above the urethra. Following a 20 mg dose of propofol for sedation, bedside ultrasound revealed a small bladder volume and a bright shadow in the anterior wall of the bladder, indicating the presence of the urinary catheter's front end and confirming bladder spasm. It was decided to administer antispasmodic drugs. After an intramuscular injection of 0.7 mg of anisodamine, the child fell asleep quietly and did not complain of any discomfort upon awakening. He was transferred back to the ward approximately 1.9 hours later, and none of the aforementioned symptoms reoccurred.

Discussion

The bladder is innervated by parasympathetic, sympathetic, sensory, and somatomotor nerves [4], as shown in **Fig-1**. Parasympathetic preganglionic fibers originating from S2~4 form the pelvic nerve, which induces detrusor muscle contraction and internal sphincter relaxation, facilitating urination. Sympathetic preganglionic fibers derived from T11-12 and L1-2 relax the detrusor muscle, promote contraction of the bladder triangle and bladder sphincter, and inhibit urination. Sensory nerve fibers play a role in conveying bladder distension and pain sensations, with some afferent fibers connected to the caudal pulp through the pelvic nerve and others connected to the lumbar pulp through the sympathetic nerve, primarily transmitting pain signals. Bladder spasm occurs due to increased mucus secretion from the urethra and bladder epithelium, resulting in an increase in C-type afferent fibers to the spinal cord. This leads to acetylcholine release from nerve endings, activation of muscarinic receptors, and involuntary contraction of the detrusor and bladder sphincter (mediated by them).

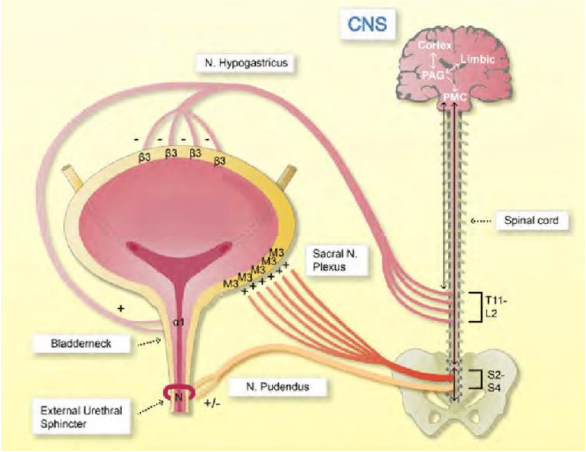

Fig-1: Schematic Figure, Neural Control of the Urinary Bladder

The bladder, bladder neck and urethra (external urethral sphincter; EUS) are innervated with parasympathic (Sarcral N. Plexus and N. Pudendus) and sympathic (N. Hypogasticus) nerves (quote from a paper published by Janssen [4]).

Caudal block is frequently used in lower abdominal and perineal surgery in children and anal and rectal surgery in adults [5]. The sacral spinal nerve supplies the visceral innervation to the penis. Caudal block is the most commonly employed intraspinal block technique in children, providing blockage to all sacral regions. As a result, it can be used for surgical analgesia in children with hypospadias. Bladder spasm can be caused by catheter stimulation of the bladder wall, urethral burning sensation, pubic arch and urethral pain, surgical trauma, or residual sympathetic innervation. Despite the administration of caudal block during anesthesia, why did this child still experience bladder spasm?

Commonly used local anesthetics for caudal block are 0.125-0.25% bupivacaine or 0.2% ropivacaine, typically at a dosage of 0.5-1 ml/kg [6]. The extent of the block is directly proportional to the volume of the local anesthetic. Studies have shown that at the same total dose, a higher volume and lower concentration result in a wider block plane and longer block duration compared to a lower volume and higher concentration. With a dose of 0.5 ml/kg of local anesthetics, the block level reaches S1, while at 0.75 ml/kg, it reaches T12,

and at 1 ml/kg, it extends to T8 [7]. The effect of ropivacaine can last from 1.5 to 12 hours. In this patient, the surgery lasted for 1.2 hours, and the effect of the caudal block remained. However, the amount of caudal block medication administered was less than 0.5 ml/kg, and the block plane may have been limited to the sacrum. Although the intraoperative analgesic effect was significant, the sympathetic nerves responsible for the bladder triangle and urethral sphincter, originating from the thoracolumbar segment, were not blocked. Therefore, the bladder and urethral sphincter continued to contract upon urethral stimulation, leading to postoperative bladder spasm in children.

Conclusions

For children with hypospadias, caudal block not only has a good analgesic effect, intraoperative opioid use reduction, and smooth vital signs but can also effectively reduce postoperative bladder spasm, suggesting that a local anaesthetic drug dose of 1 ml/kg, for a total of not more than 20 ml, makes the block plane at least T11 to avoid the occurrence of postoperative bladder spasm and increase the comfort of children.

Patient Consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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