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Case Report

Combined Use of Epidural Analgesia and Intravenous Lignocaine for Enhanced Recovery in a Tetraplegic Patient with Autonomic Hyperreflexia: a Case Report

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ABSTRACT

A 50-year old man with a complete spinal cord section at the C6-C7 level and suspected Autonomic Hyperreflexia (AH) was scheduled for cystectomy with urinary diversion. In addition to general anesthesia, both thoracic epidural analgesia and intravenous lignocaine were used to prevent AH upon surgical stimulation and facilitate the postoperative recovery of bowel function. The patient remained remarkably stable throughout the surgery. He made a good postoperative recovery and started eating at day 3. The theoretical advantages of epidural analgesia and intravenous lignocaine for the management of patients with spinal cord injuries complicated by autonomic hyperreflexia are briefly reviewed.

Keywords: Spinal cord injury; Anesthesia; Autonomic hyperreflexia; Epidural; Lignocaine

INTRODUCTION

Urological surgery is commonly required in patients with spinal cord injuries. The anesthetic management of these patients poses specific challenges. Autonomic Hyperreflexia (AH) is frequent in patients with complete spinal cord lesion above T6 [1]. In patients suffering AH, surgical stimulation can trigger uncontrolled sympathetic activation leading to potentially life-threatening hypertension and cardiac arrhythmias. Postoperative ileus can also be particularly prolonged and negatively impact on postoperative recovery in quadriplegic patients undergoing abdominal surgery [2]. We report a case of combined use of intravenous lignocaine and epidural analgesia in a quadriplegic patient with suspected AH undergoing cystectomy with urinary diversion.

CASE PRESENTATION

A 50-year old man with a complete spinal cord section at the C6-C7 level was scheduled for cystectomy with urinary diversion. The patient's weight and height were 68 kg and 170 cm, respectively. He had been quadriplegic for three years following a traffic road accident. Ten days before surgery, during a flexible cystoscopy performed without anesthesia, he suffered from a severe headache associated with an elevation of the systolic blood pressure up to 195 mmHg leading to the diagnosis of AH.

On the day of surgery, the patient was admitted to the theater and monitored with a 5-lead EKG, a non-invasive blood pressure cuff, and a pulse oximeter. An epidural catheter was inserted at the T10-T11 level under local anesthesia. General anesthesia was then induced with sufentanil 10 µg, midazomal 2 mg, lignocaine 100 mg, and propofol 60 mg. Full muscle relaxation was achieved with an intravenous bolus of 70 mg of rocuronium before the trachea was intubated. Sevoflurane in an air:oxyen mixture was used for the maintenance of anesthesia. The end-tidal concentration of sevoflurane was adjusted to keep the entropy of the electroencephalogram (M-Entropy, Acertys-GE, Madison, WI) between 40 and 60. An arterial line and a central venous catheter were inserted after induction of general anesthesia. A bolus of 5 ml of ropivacaine 0.2% was administered epidurally before skin incision and followed by a continuous infusion of 5 ml/h. A continuous intravenous infusion of lignocaine 2% added with 1 mg/ml of ketamine was also started and continued until the end of surgery. In addition, a bolus of 50 mg/kg of magnesium was administered over an hour just after induction of anesthesia.

The surgical procedure lasted three hours and was uneventful. No episode of hypertension or bradycardia suggestive of AH occurred. A total of three boluses of ephedrine 9 mg were administered to correct transient episodes of low blood pressure. No anti-hypertensive medication or additional bolus of sufentanil were used.

The patient was extubated in the operating room shortly after skin closure and was discharged from the recovery unit six hours after the end of surgery. One gram of intravenous paracetamol was given every 6 hours and the epidural infusion of ropivacaine 0.2% was maintained at 5 ml/h until the morning of the fourth postoperative day. After withdrawal of the epidural catheter, an intravenous infusion of lignocaine 2% was restarted for 48 hours at a rate of 4.5 ml/h (1.33 mg/kg/h).

The patient remained perfectly comfortable during the whole postoperative period with a numeric rating scale for pain constantly reported at 0 out of 10. Upon physical examination, bowel movements were audible as of the first postoperative day. The nasogastric tube was removed on the morning of the second postoperative day and the patient started eating on the third postoperative day. His recovery was unfortunately complicated by a urinary infection requiring 10 days of intravenous antibiotics. He was finally discharged from hospital at postoperative day 20.

DISCUSSION

Patients with SCI are vulnerable to several types of postoperative complications including cardiovascular instability, respiratory insufficiency and ileus [2]. A good understanding of the pathophysiology of chronic SCI and its implications during the perioperative period is therefore essential to anesthesiologists dealing with these patients. AH is a potentially life-threatening complication of chronic Spinal Cord Injuries (SCI). Its prevalence is highest in patients with complete spinal cord lesions above the T6 level in whom it ranges between 48% and 85% [3,4]. The most common sign of AH is an elevation of systolic blood pressure triggered by a noxious or a non-noxious stimulation below the level of the lesion [3]. These episodes of hypertension may be accompanied by headache, profuse sweating, head and neck rash, and changes in heart rate most commonly consisting in tachycardia. Hypertension results from the uncontrolled sympathetic activation and intense vasoconstriction below the level of the spinal cord lesion while flush and headaches are explained by a reflex activation of the parasympathetic nervous system causing vasodilation [5]. Diagnostic criteria include an increase in systolic blood pressure of at least 20 mmHg above the baseline. However, levels of systolic blood pressure as high as 250-300 mmHg have been reported in patients suffering AH [1]. Avoiding these surges in blood pressure upon surgical stimulation is therefore essential to prevent potentially life-threatening complication such as cerebral hemorrhage [6].

Our patient previously had an uneventful surgery for pressure sore defect. This could have been falsely reassuring regarding the risk of AH. However, lower urinary tract stimulations such as cystoscopies [7] and visceral surgery [3] are among the most frequent triggers of

AH in susceptible patients and often reveal the diagnosis. The fact that the patient presented signs and symptoms suggestive of AH during a flexible cystoscopy performed without anesthesia a few days before surgery lead us to consider him at high risk of perioperative AH. Several anesthetic techniques have been proposed to block the sympathetic activation during surgery and prevent AH in susceptible patients. Modern volatile anesthetics such as sevoflurane have both vasodilatory properties and the ability to mitigate sympathetic reflexes. In patients undergoing transurethral litholapaxy the EC95% of the end-tidal concentration of sevoflurane in a 50% nitrous oxide:oxygen mixture to block AH was 3.8% [8]. This concentration can be reduced by 30% by administering remifentanil at a target-controlled concentration of 3 ng/mL [9]. Neuraxial blockade using local anesthetics is another effective way to block the sympathetic outflow and thereby to attenuate intraoperative AH [10]. Spinal anesthesia has been successfully used during urological surgery [11] and cesarean delivery [12]. Its use is associated with a remarkable cardiovascular stability and, although technical difficulties may occur, spinal anesthesia seems feasible in the vast majority of cases [11]. The use of epidural blockade has also been reported though less commonly [13]. Technical difficulties and block failures could be more common than for spinal anesthesia because of spinal deformations and distortion of the epidural space. In contrast to single shot spinal anesthesia, epidural infusions can be continued postoperatively to facilitate postoperative pain control, reduce opioid requirements, and hasten postoperative recovery [10]. Although there is no evidence to support it, postoperative epidural infusions of local anesthetics could theoretically help prevent visceral pain and AH associated with the return of bowel function. Intravenous lignocaine is increasingly used during surgery as component of multimodal analgesia. It also contributes to attenuate the sympathetic response to laryngoscopy [14] and has recently been used for the treatment of AH [15]. In addition, intravenous lignocaine accelerates the recovery of bowel function after abdominal surgery [16]. Finally, magnesium is another frequently used anesthetic adjunct. It decreases systemic vascular resistances and reduces catecholamine-induced hypertension in addition to its anti-NMDA properties [17]. Magnesium has also been successfully used to treat AH [18,19].

The place of epidural analgesia during surgery has been decreasing steadily over the last years with the advent of minimally invasive surgery and the development of multimodal analgesia regimens [20]. We however believe that epidural analgesia still has a role in specific patient's populations such as patients with SCI. It has indeed a unique ability to block both sensory pathways and sympathetic outflow, which is of particular value regarding the prevention of AH. It also facilitates the postoperative recovery of bowel function by its effect on the sympathetic/parasympathetic balance [10].

Intravenous lignocaine has several effect that may, at least theoretically, potentiate the advantages of epidural analgesia. Firstly, the perioperative use of intravenous lignocaine has a sparing effect on general anesthetic requirements [21]. Secondly, lignocaine has analgesic and anti-hyperalgesic properties that reduce opioid requirements and opioid related side-effects [16]. Finally, thanks to its anti-inflammatory properties and to a direct excitatory effect on the intestinal smooth muscles, lignocaine helps shorten the duration of postoperative ileus [22]. Combining several local-anesthetic based techniques such as epidural analgesia and intravenous lignocaine infusion raises the question of systematic toxicity. The doses of lignocaine used can result in plasma concentrations up to 4 mg/L [23]. Although this remains far below the threshold of systemic toxicity, the infusion of intravenous lignocaine was discontinued at the end of surgery and only restarted after the removal of the epidural catheter.

CONCLUSION

In conclusion, this is the first report of a combined use of intravenous lignocaine and epidural analgesia. This approach proved particularly successful for our patient and potentially deserves further investigations. The relative contribution of epidural analgesia and intravenous lignocaine to the patient outcome can however not be determined.

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