

SYSTEMIC TOXICITY REACTIONS DUE TO EPIDURAL CATHETER DISPLACEMENT

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Abstract- Toxic reactions to local anesthetics may occur due to over dose, accidental intravenous injections or rapid systemic uptake of the drugs. But there have been few reports on toxicity due to displacement of epidural catheters after an initial correct insertion and performance of a test dose. Here we present a case of local anesthetics toxicity due to possible displacement of the epidural catheter during APR (Ant-post Colporrhaphy) surgery. The patient's level of consciousness decreased at the end of surgery. She demonstrated CNS and cardiovascular toxicity due to local anesthetics. Consequently the patient was intubated and transferred to ICU, where she received mechanical ventilation along with other supportive treatments such as inotropic and antiepileptic drugs. We concluded that supportive therapy is the treatment of choice for such cases.

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Key words: Epidural catheter, toxic reaction, local anesthetics

INTRODUCTION

Local anesthetics are relatively safe drugs if administered in appropriate dosage and at the correct anatomic location. Bupivacaine, lidocaine and tetracaine are the local anesthetics more often used in regional anesthesia and peripheral nerve blocks nowadays. Bupivacaine is an amidic, potent local anesthetic, which possess the longest action of duration among local anesthetics. It is enzymatically destructed slower than lidocaine in liver. Excretion of amidic metabolites is through renal system. CNS is more prone to local anesthetic toxicity than the cardiovascular system and lower levels of local anesthetic in blood will initiate CNS toxicity (1). Bupivacaine toxicity is an exception to this rule *i.e.* cardiotoxicity occurs almost simultaneously to CNS manifestations, yielding a smaller margin of safety.

The main concern in epidural anesthetic procedures is reassurance of the correct insertion of the catheter. By applying the "loss of resistance" technique through L3-L4 or L4-L5 spaces, the epidural space is determined. A small dose of lidocaine 2% (3 ml) plus epinephrine is injected to test the correct insertion of the catheter and after gaining reassurance, the catheter is fixed and local anesthetic injected.

Here we present a case of local anesthetics toxicity due to possible displacement of the epidural catheter during APR (Ant-Post colporrhaphy) surgery.

CASE REPORT

The patient was a 60-yr-old, 80 kg, 150 cm, female (BMI: 35.5) with a history of urine-stool incontinency following ten vaginal deliveries who was a candidate for an Ant-Post colporrhaphy surgery. She had a history of asthma and bronchiectasia.

In preoperative evaluation, mild exertional dyspnea and end expiratory wheezing at the base of both lungs were noticed. The patient was not cooperative for pulmonary function test.

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Echocardiography revealed mild TR, mild MR and an EF of 55%. Upon arrival to the operating room the standard monitoring were applied. The vital signs were checked and recorded (BP: 120/75 mm Hg, PR: 80 bpm, RR: 16 pm).

The patient was hydrated with Ringer Lactate solution and received supplementary oxygen. The epidural space at L4-L5 level was found by administering the "loss of resistance" technique and an epidural catheter was inserted and rechecked for correct position through a test dose (3 ml of Lidocaine 2% + 15 µl of Epinephrine 1/200000). No changes in pulse rate or blood pressure or sacral sensation were detected after 3 minutes. So 15 ml of bupivacaine 0.5% was injected slowly. At this point the catheter was covered with sterile gauzes and fixed to the body. The surgery team then removed the patient and placed her in lithotomy position and the operation started. Twenty minutes later her pulse rate dropped to 50 bpm without any changes in blood pressure. Administration of 0.5 mg atropine terminated the bradycardia. The sensory block level was at T6 dermatome. No additional local anesthetics were injected in the epidural catheter; also the patient did not receive any sedative or narcotic drugs during the surgical procedure which took 1.5 hours.

At the end of the operation the patient became disoriented and couldn't answer the questions. No considerable change in vital signs was noticed. The patient was transferred to the recovery room and received supplementary oxygen. Thirty minutes later, her level of consciousness was severely decreased and she didn't show any reaction to painful stimulation and had shallow respiration. She was then promptly intubated by administering 300 mg of thiopental sodium (4 mg/kg) and succinylcholine 120 mg (1.5 mg/kg). The ABG simultaneously revealed severe respiratory acidosis ($P_a\text{CO}_2$: 110 mmHg, pH: 6.91, Bicarbonate: 21.8 meq/l, $P_a\text{O}_2$: 215 mmHg). The patient's BP dropped to 80/50 mmHg one hour after intubation and did not increase despite receiving 1500cc of Ringer lactate solution. The blood pressure was maintained at the level of 100/65 mmHg by administering 5 µg/kg/min of dopamine. She was then transferred to the ICU. Upon arrival, the patient developed generalized tonic

colonic seizure which was ceased by IV injection of 10 mg of diazepam and then she received a loading dose of phenytoin (15 mg/kg) followed by a maintenance dose of phenytoin (5 mg/kg) in three divided doses. No ECG changes were noted. The pulse rate remained regular throughout the episode of seizure without any evidence of cardiac arrhythmias. Laboratory test consisting of blood sugar and blood electrolytes were normal. Neurological consultation reported no pathology on physical examination, CT scan and EEG. Gradually she was fully recovered with no residual complications. On arrival to ICU she received mechanical ventilation under CMV mode which was changed to SIMV plus pressure support gradually as the patient regained spontaneous breathing. During the next two days the mode of ventilation was changed to CPAP. All of the ABG tests after intubation were normal without any increase in $P_a\text{CO}_2$.

The patient was extubated 3 days post intubation and phenytoin was discontinued after 5 days on the basis of normal CT and EEG results and no recurrence of seizure. She discharged on the 7th postoperative day with stable conditions.

DISCUSSION

In patients with respiratory disease who require low sensory block level, regional anesthesia is safer than general anesthesia. Local anesthetics could have potential complications such as neurotoxicity, allergic and systemic toxicity reactions (2).

The initial symptoms of CNS toxicity are feeling of light headedness and dizziness frequently followed by visual and auditory disturbances. Objective signs are usually excitatory including shivering, tremor and muscle twitching. Ultimately generalized convulsion of a tonic-colonic type occurs. In the final stages of local anesthetic toxicity, cardiac manifestations will predominate *e.g.* hypotension, bradycardia and cardiac arrest. There are several reports on bupivacaine systemic toxicity after continuous cervical epidural infusion (3), intra-articular injection and orbital indwelling catheter gaining entrance to subarachnoid space (4, 5). Our patient received 15 ml of 0.5% Bupivacaine via

epidural catheter and sensory block level never raised above the T6 dermatome. As the blood pressure remained stable throughout the operation, complications can not be explained by high block in which the manifestations apart from hypotension consist of upper extremity weakness, sensory loss, diminished respiratory efforts and difficulty in speaking. Fortunately the cervical spinal cord segments supplying the diaphragm are relatively resistant to the motor blocking effect of epidural local anesthetics and in most cases, respiratory function remains adequate making endotracheal intubation unnecessary (6). In this patient we encountered some manifestations such as dizziness, disorientation, disability to make verbal contact, decreasing respiratory rate and convulsion that indicated systemic toxicity. Measuring the blood level of bupivacaine is a good assistance in making a definite diagnosis which unfortunately we are not able to determine here. Therapeutic management in this patient consisted of tracheal intubation to support ventilation, administration of dopamine to maintain blood pressure and antiepileptic drugs to treat and prevent recurrence of seizure along with other supportive managements. We believe that the epidural catheter was dislocated while placing this patient in the lithotomy position, consequently the epidural vessels were injured and systemic uptake of local anesthetic took place which caused systemic toxicity manifestations. To prevent displacement of the catheter we recommend suturing it to the skin and taking extreme care while changing the patient's position.

Although the incidence of seizure is low (2 per 1000) after peripheral blocks using local anesthetics (7), the anesthesiologist should always keep in mind the possibility of local anesthetic systemic uptake due to dislocation of the catheter, which may cause a variety of systemic toxic manifestations.

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