Fatal Peripartum Cardiomyopathy after Bupivacaine Local Injection in Elective Cesarean Section: A Case Report

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Bupivacaine is frequently used for pain control and local anesthesia. However, it is associated with certain acute and fatal side effects, although rare, including cardiac and central nervous system toxicities. In particular, bupivacaine-induced cardiac toxicity may be fatal. This condition can be diagnosed as bupivacaine-induced cardiotoxicity by excluding other causes and determining a history of bupivacaine administration. However, in emergency situations, recognizing bupivacaine toxicity can be difficult due to the physician’s lack of awareness regarding the condition or in the absence of clear communication regarding the patient’s medical history. In the current case report, we describe our experience with strong suspected bupivacaine-induced cardiotoxicity in a patient who underwent cesarean section along with a review of the literature.

Key Words: Bupivacaine, Cardiomyopathy, Lipid rescue

Introduction

Bupivacaine is a long-acting, local anesthetic agent that is widely used for cutaneous infiltration, intra-articular injection, peripheral nerve blocks, epidural anesthesia, and spinal anesthesia. However, the use of bupivacaine is associated with rare but fatal adverse effects, such as central nervous system (CNS) and cardiovascular toxicity, caused by accidental intravascular injection or excessive systemic resorption. Such cardiotoxicity can manifest as cardiac depression, ventricular tachycardia, asystole, and/or electromechanical dissociation. A history of bupivacaine use and exclusion of differential diagnoses is crucial for diagnosing this condition. A dramatic response to 20% lipid rescue infusion is well known. However, it is difficult to recognize bupivacaine toxicity in the absence of precise history taking or lack of awareness of this disease entity.

Here, we present a case of strong suspected bupivacaine-induced peripartum cardiomyopathy (PPCM) after cesarean section with short review.

Case Report

A 28-year-old primi-gravida woman was admitted to our hospital for dyspnea immediately following delivery by cesarean section in another clinic. The patient had no history of illness, and all of her medical condition including vital signs were normal prior to the cesarean section procedure. An elective cesarean section was performed under combined spinal-epidural anesthesia using bupivacaine 0.5% and fentanyl anesthesia. Spinal anesthesia was performed at the L3-4 intervertebral level using a 23 G spinal needle. Once clear cerebrospinal fluid was confirmed, 9 mg bupivacaine and 15 μg fentanyl was injected immediately. The procedure was uneventful until the occurrence of dyspnea, when peripheral oxygen saturation (SpO2) decreased to 91% despite oxygen supplementation. The patient did not receive any kinds of cardiotoxic drug during procedure.

When the patient arrived in the emergency room, the chief complaint was severe dyspnea. In the emergency room, the initial blood pressure was 90/70 mm Hg, pulse rate was 98 beats/min, respiratory rate was 32 breaths/min, and body temperature was 36.5°C. The patient was alert, and active bleeding, petechiae, or abnormal leg swelling was not observed. Chest auscultation...
tion revealed coarse crackles in both lung fields. The patient’s initial electrocardiogram (EKG) showed sinus tachycardia and mild ST segment elevation in the V1–V3 leads (Fig. 1A). After 3 hours, the EKG showed a change to supraventricular tachycardia (Fig. 1B). Laboratory findings were normal, except for mild leuko-

Fig. 1. (A) Initially, the electrocardiogram showed sinus tachycardia and mild ST segment in the V1–V3 leads in the emergency room. (B) After 3 h, electrocardiogram changed to supraventricular tachycardia. (C) On 5th admission day, electrocardiogram showed normal findings.

Fig. 2. (A) Chest radiography showed bat-wing consolidation in both lungs. (B) Chest computed tomography (CT) shows consolidation, ground-glass opacity, interlobular septal thickening, and pleural effusion in both lungs.
cytosis (white blood cell count, 19,880/mm³) and cardiac biomarker elevation. Cardiac biomarkers were significantly increased as follows: creatine phosphokinase (CPK) level: 381 IU/L (reference range<190 IU/L), creatine kinase-MB (CK-MB) level: 11.45 ng/mL (reference range<4.94 ng/mL), troponin-T level: 0.314 ng/mL (reference range<0.1 ng/mL), and pro-brain natriuretic peptide (BNP) level: 7,412 pg/mL (reference range<115 pg/mL). Severe hypoxemia (PaO₂: 49 mm Hg) was noted in arterial blood gas analysis during ventilation at 5 L/min via nasal prong. Chest radiography showed bat-wing consolidation in both lung fields (Fig. 2A). Chest computed tomography (CT) revealed consolidation, ground-glass opacity, interlobular septal thickening, and

Fig. 3. (A) The initial echocardiography revealed dilated left ventricular cavity (56 mm) with severe left ventricular global hypokinesia with a decreased ejection fraction of 31%. (B) Follow-up echocardiography demonstrated full recovery, with an increased ejection fraction of 58%.
pleural effusion in both lungs (Fig. 2B). This finding is in accordance with pulmonary edema. No evidence of pulmonary thromboembolism was noted in computed tomography scans of the chest. Echocardiography showed a dilated left ventricular (LV) cavity (56 mm) and severe left ventricular global hypokinesis with an ejection fraction (EF) of 31% (Fig. 3A). Next, we performed coronary angiography, which revealed normal coronary arteries findings. The patient had no known etiology of peripartum heart failure, and PCCM was thus diagnosed. She was treated with 13.3 \( \mu \text{g/kg/min} \) dopamine, low-molecular-weight heparin 40 mg, digoxin 0.125 mg, intermittent furosemide intravenous injection, aspirin and ventilator care along with the placement of an intra-aortic balloon pump to correct refractory hypotension. Fortunately, the patient’s clinical parameters improved after the 3rd admission day. On the 5th admission day, the patient showed signs of much improvement, with EKG normalization (Fig. 1C); a history of bupivacaine usage during the cesarean section procedure was subsequently elicited from the local clinic where cesarean section was performed. We finally diagnosed the condition as strong suspected bupivacaine-induced PPCM, because we did not find out the other etiologies responsible for this event. Follow-up echocardiography revealed an increased EF of 58% on the 10th admission day (Fig. 3B), and she was subsequently discharged without any sequelae on the 16th admission day.

**Discussion**

Bupivacaine is widely used as a long-acting local anesthetic agent; however, acute and fatal side effect of local anesthetics usually occur simultaneously after an overdose of local anesthetics or accidental intraveacular injections. Systemic toxicity from local anesthetics may occur in as many as 1:1000 peripheral nerve blocks. Cardiac toxicity is one of the adverse effects of bupivacaine; it may variously manifest as cardiac depression, ventricular tachycardia, asystole, and/or electromechanical dissociation.

The mechanism of cardiotoxicity is explained by bupivacaine’s interaction with cardiac sodium channels, disruption of arterioventricular nodal conduction, depression of myocardial contractility, and indirect effects mediated by the central nervous system.

Bupivacaine is also the most commonly used local anesthetic in epidural anesthesia during labor, as well as in postoperative pain management. However, unfortunately, obstetric patients could also experience life-threatening bupivacaine-induced cardiotoxicity, as in our present case. With respect to the pathogenesis in our case, we consider that a small amount of bupivacaine leaked into the blood vessels, and a similar case had been reported in 2007. Although the blood levels of the anesthetic agent can be possibly measured for accurate diagnosis, this may not provide a confirmative diagnosis of the causative factor of cardiotoxicity since it may not correlate with the toxicity; moreover, the results of such analysis are typically not obtained rapidly, i.e., in a clinically useful time. Therefore, diagnosis should be guided by clinical presentation. In the present case, we could suggest this patient could be caused by bupivacaine. Because she has no history of medical illness, all vital signs were stable preoperatively, no known etiology of PPCM, and she has no any sequelae at discharge time.

Rosenblatt et al reported the usefulness of 20% lipid emulsion therapy for resuscitating a patient after presumed bupivacaine-related cardiac arrest for the first time. Subsequently, several additional reports have been presented about successful lipid rescue in similar cases. Although the 20% lipid emulsion therapy is now standard therapy in bupivacaine-related cardiac arrest, the precise mechanism of action remains unknown. At first, since the history of bupivacaine use for epidural anesthesia was not available, we did not recognize the condition as bupivacaine-induced PPCM. Therefore, we only administered standard intensive care unit (ICU) care without 20% lipid emulsion therapy. If bupivacaine-induced PPCM had been recognized earlier, and lipid rescue therapy had been administered, we believe that the patient’s clinical course would have improved more rapidly.

Although lipid rescue treatment of bupivacaine-induced cardiac toxicity is well described in the literature, its potential lifesaving impact is not widely recognized by clinicians in the emergency room and intensive care unit in Korea. It is our hope that this article will stimulate recognition of bupivacaine-related peripartum cardiomyopathy.
REFERENCES


