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PROFOUND PROLONGED BRADYCARDIA AND HYPOTENSION AFTER INTERSCALENE BRACHIAL PLEXUS BLOCK WITH BUPIVACAINE

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Abstract—Background: Interscalene brachial plexus blocks have been a routinely performed method of anesthesia for shoulder surgery that decreases the need for general anesthesia, length of stay, and recovery time. We describe a case of bupivacaine toxicity after an interscalene block. **Case Report:** The patient was a 66-year-old man who presented to our Emergency Department by emergency medical services from an ambulatory surgery center where he had undergone rotator cuff surgery, with bradycardia and hypotension. His symptoms began upon completion of the surgery in which he received interscalene nerve block with bupivacaine and lidocaine. He was given three doses of 0.5 mg atropine and one dose of 1 mg epinephrine for a heart rate of 40 beats/min without any improvement prior to arrival. His bradycardia was refractory to atropine. He was started on a dopamine drip and transferred to the coronary care unit. The timing of his symptoms, minutes after his regional nerve block, and his complete recovery with only supportive care, make the diagnosis of bupivacaine toxicity likely. **Why Should an Emergency Physician Be Aware of This?:** Despite the safety profile of local anesthetics, we must be aware of their potential side effects. Whereas most adverse reactions are secondary to misdirection of anesthetic or accidental vascular puncture, local anesthetic systemic toxicity (LAST) is the major cause of significant adverse events with regional anesthesia. As regional anesthesia becomes more common, emergency physicians must be more aware of the potential complications and be able to both diagnose and treat. © 2017 Elsevier Inc. All rights reserved.

Keywords—regional anesthesia; bradycardia; bupivacaine

INTRODUCTION

Interscalene brachial plexus blocks have been a routinely performed method of anesthesia for shoulder surgery. It is performed by injecting a large volume of local anesthetic within the interscalene groove in the lower neck to accomplish blockade of the brachial plexus, resulting in anesthesia of the shoulder, arm, and elbow. It decreases the need for general anesthesia, hospital length of stay, and recovery time (1). Complications, including phrenic nerve paralysis, pneumothorax, and hematoma formation are generally rare. However, there have been a few reported cases of asystolic cardiac arrest associated with interscalene brachial plexus blocks (1).

CASE REPORT

The patient is a 66-year-old man who presented to our Emergency Department (ED) by emergency medical services from an outpatient ambulatory surgery center where he had undergone elective left rotator cuff surgery with a chief complaint of bradycardia and hypotension. His past medical history was significant for diabetes, hypothyroidism, and sleep apnea. Past surgical history was significant for coronary artery bypass grafting 11 years prior and lobectomy from lung cancer 30 years prior. His symptoms began upon completion of the surgery and he was given three doses of

0.5 mg atropine and one dose of 1 mg epinephrine for a heart rate of 40 beats/min without any improvement prior to arrival. As per ambulatory care center notes, he was given propofol for sedation and underwent an ultrasound-guided interscalene nerve block with 30 mL of 0.5% bupivacaine and 10 mL of 2% lidocaine. His initial heart rate was 40 beats/min, blood pressure was 130/100 mm Hg, rectal temp was 36.5°C (97.7°F), and finger stick glucose 210 mg/dL. His SpO₂ was 92% on 4 L O₂ by nasal cannula and his respiratory rate was 18 breaths/min. His physical examination was baseline unremarkable other than mottled skin. His electrocardiogram showed sinus bradycardia, with a rate of 38 beats/min with PR prolongation and T-wave inversions in V1 and aVL that were new since his preoperative electrocardiogram. He denied chest pain but endorsed dyspnea. Soon after arrival to the ED, his blood pressure dropped to 80/40 mm Hg and his mental status declined, necessitating intubation for airway protection. Again the bradycardia was refractory to atropine. He was started on a dopamine drip and transferred to the coronary care unit. Bedside ED ultrasound showed no global hypokinesis, no pericardial effusion, and no evidence of right heart strain. Computed tomography angiogram was performed to exclude pulmonary embolism. His initial laboratory results were significant for a lactate of 10.1 mmol/L, which decreased to 3.6 mmol/L on the subsequent blood draw 40 min later, and a serum magnesium of 0.8 mg/dL, which was repleted. The next day his thyroid-stimulating hormone test resulted as normal at 2.01 uIU/mL. In < 4 h, his hemodynamic status normalized. He was weaned off the ventilator the next day and monitored for events on telemetry. He recovered without incident and was discharged home in several days. Because this patient had multiple other comorbidities, it was challenging to ascribe his symptoms solely to bupivacaine on initial presentation. However, the timing of his symptoms, minutes after his regional nerve block, and his complete recovery with only supportive care, make the diagnosis of bupivacaine toxicity much more likely.

DISCUSSION

Bupivacaine, a long-acting, lipid-soluble local anesthetic, has many useful applications and its toxicity is relatively uncommon. Rarely, it has been reported to produce transient cardiac dysrhythmias, most commonly bradycardia. First synthesized in 1957, cardiotoxicity attributable to bupivacaine was first documented in 1969 in 23 cases of fetal death when used in obstetrics for paracervical block to treat labor pain (2). Most other adverse events were largely confined

to central nervous system toxicity. Many authors originally reasoned that convulsions may have led to hypoxia, which caused cardiovascular collapse (2). When hypotension, bradycardia, and cardiac arrest were reported, it had been attributed to central neuraxial blockade (subarachnoid injection) (2). However, by 1983, well-documented cases of cardiac arrest associated with bupivacaine use led to the eventual discovery of the potential use of lipid emulsion, or Intralipid® (Baxter Healthcare Corporation, Deerfield, IL), therapy for refractory cardiac arrest (3). Typically, the use of ultrasound guidance allows for direct visualization of the injecting needle as well as the intended nerve and its surrounding vasculature. This has led to the usage of less volume as well as fewer side effects (4). Many different hypotheses have been proposed regarding the pathogenesis of hypotensive and bradycardic events after interscalene brachial plexus blocks. The first is that incidental blockage of sympathetic chain or stellate ganglion could affect autonomic influence. Another is that incidental blockage of spinal nerves to sternocleidomastoid muscles (C2–C4) could affect the carotid sinus baroreceptor reflex arc and lead to vagal stimulation. Yet another involves direct manipulation of the carotid sinus due to frequent neck turning and arm traction of shoulder surgery exacerbated by neck swelling from physical volume of local anesthetic during the interscalene block. The sitting position is frequently used in shoulder surgery because it reduces the need for heavy traction; however, this position has been shown to have an increased risk of potential complication of hypotensive or bradycardic events, with a reported incidence of 13–28% (5).

Also frequently cited in cases of cardiac collapse after interscalene blocks, especially in the sitting position, is the “Bezold-Jarisch reflex,” which is when venous pooling results in an empty but hypercontractile ventricle, which causes stimulation of intramyocardial mechanoreceptors and increases vagal tone, causing bradycardia and hypotension (1). These vagal responses would be expected to last only minutes and not hours, as it did in our patient.

It is possible that part of the solution was inadvertently injected intravascularly or intrathecally. We assume that frequent aspiration tests were performed without obtaining blood during the procedure in our patient, however, a negative aspiration test does not rule out intravascular injection. An intrathecal injection would cause complete spinal anesthesia (6). Our patient had motor function, making this an unlikely cause for his presentation. Accidental neuraxial blockade with accompanying bradycardia has been described in rare case reports (7). Ultrasound guidance of the procedure should make all of these scenarios less likely (4).

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