

Arrest Under Anesthesia - What was the Culprit? A Case Report

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Abstract

Our case describes a 70 year-old male patient with no significant cardiac history who presented for a hemicolectomy for recurrence of colon cancer. Induction of general anesthesia was uneventful. A dexmedetomidine infusion was started prior to incision and a bolus of bupivacaine was administered via an indwelling epidural catheter. Twenty minutes after surgical incision, the patient acutely developed bradycardia that progressed to asystolic arrest. CPR was immediately initiated with return of circulation after three minutes. The case was aborted and the patient made a full recovery. The patient was evaluated by cardiology postoperatively. A detailed evaluation uncovered additional pertinent history of a syncopal event 1.5 years ago, and an episode of hypotension and bradycardia more recently that was not further investigated. In this case, dexmedetomidine and bupivacaine likely functioned synergistically to exacerbate an underlying propensity for bradycardia and increased vagal tone from surgical manipulation that culminated in asystole under anesthesia.

Keywords: Anesthesia; Bradycardia; Bupivacaine; Dexmedetomidine

Introduction

As enhanced recovery programs continue to gain traction in the field of perioperative medicine, an intraoperative multimodal analgesic approach has become increasingly commonplace [1,2]. Multimodal analgesia involves the use of multiple, simultaneous and synergistic mechanisms of pain control to improve the overall analgesic effect and reduce the doses of any single agent to minimize risks of side-effects. Instead of relying mainly on opioids for perioperative pain control, anesthesiologists are now using combinations of acetaminophen, non-steroidal anti-inflammatory drugs, alpha-2-adrenergic receptor agonists, and N-Methyl-D-aspartate receptor antagonists with improved outcomes and patient satisfaction [3,4].

Case Report

A 70 year-old male patient with no significant cardiac history and a PMH of hyperlipidemia, BPH, GERD, childhood seizures, and metastatic colon cancer s/p chemotherapy and resection in 2010, portal vein embolization for liver metastases in 2014, and right hepatectomy in 2015, presented to the OR for a hemicolectomy and debulking for tumor recurrence.

Anesthesia induction was uneventful. The patient received 2 mg of midazolam, 180 mg of propofol, 50 mg of rocuronium for induction of general anesthesia. The airway was secured with a 7.5 standard cuffed endotracheal tube. Dexmedetomidine infusion was started at 0.4 mcg/kg/hr prior to incision and 20 mg of bupivacaine (0.25%) was bolused via the epidural 5 minutes before incision. The epidural

infusion of bupivacaine (0.05%) and hydromorphone (8 mcg/ml) was initiated at a rate of 6 ml/hr.

Twenty minutes after surgical incision was made, the patient acutely developed bradycardia that progressed into asystolic arrest within 90 seconds. A code was called and CPR was immediately initiated. 1mg of epinephrine was administered with return of circulation after 3 minutes.

A combined decision by the surgical and anesthesia services was made to abort the procedure and the cardiology service was called to evaluate the patient in the PACU. Upon detailed evaluation, additional history was elicited that revealed a syncopal episode that occurred 18 months previously and for which there was no further workup. The patient also endorsed an episode of lethargy and poor responsiveness a few months ago that prompted emergency medical assistance. He was found to be hypotensive and bradycardic but recovered spontaneously and patient attributed this to a vagal episode.

The patient continued to recover uneventfully in the PACU after he was seen by cardiology. Repeat EKG and Echocardiography showed no abnormalities. Troponin and all other laboratory values were negative and the patient was transferred to a telemetry floor the next day. A follow-up nuclear stress test showed no evidence for stress-induced ischemia or infarct.

Discussion

In this case, if the patient's history of previous syncope and bradycardia had been documented, the patient might have been referred for a cardiology consultation prior to surgery.

The patient had multiple prior uneventful general anesthetics. However, dexmedetomidine and epidural bupivacaine had not previously been administered.

Bupivacaine toxicity causing arrhythmias and cardiac arrest is well described in the literature. While bradycardia and asystole is more commonly reported under spinal anesthesia, reports of these events under epidural anesthesia have also been described [5-7]. Most theories regarding bradycardia and asystole during neuraxial anesthesia implicate the direct effects of bupivacaine on the heart. Still other possible mechanisms include inhibition of sympathetic efferents and decreased venous return which in turn activates reflexes that can cause bradycardia. At least three such reflexes have been proposed and the effector arm of each of these results in increased vagal tone. A high level of sympathetic blockade may alter the balance of autonomic input to the heart, thereby favoring vagal tone, and bradycardia [5,8].

Dexmedetomidine can cause a biphasic cardiovascular response, where an initial bolus causes an increase in blood pressure followed by a reflex decrease in heart rate and subsequent decline in blood pressure during continuous infusion. Studies have shown that both sinus and atrioventricular nodal functions can be depressed by dexmedetomidine; there have been a few case reports describing dexmedetomidine-induced bradycardia culminating in cardiac arrest [9,10].

In addition to drug effects, bradycardia during general anesthesia can be attributed to triggering of the vagal reflex by surgical manipulation [11]. This reflex bradycardia during surgery is well-described in medical literature, with various studies demonstrating an average decrease in blood pressure of 20 mmHg during surgical manipulation of abdominal contents [12,13].

This case highlights the potential for compounding of adverse effects when using multimodal therapy. But equally important, this case

underscores the importance of a focused yet detailed anesthesia preoperative evaluation that may reveal critically important information [14].

Conclusion

It was initially presumed that this patient probably had an undiagnosed underlying sinus node dysfunction. Combined with intraoperative administration of drugs associated with bradycardia) and surgical manipulation triggering increased vagal tone, these factors likely interacted synergistically to precipitate bradycardia and asystole during surgery. During his postoperative recovery on telemetry, the patient had an observed event of non-sustained bradycardia, without evidence of heart block.

Cardiac Electrophysiology consultation did not recommend additional EP studies in the absence of structural heart disease, ischemic disease, and conduction system disease. It was concluded that the patient had a profound vagal event potentiated by anesthesia and medications. Their recommendation was preoperative admission and placement of temporary pacing wire prior to rescheduled procedure as well prophylactic atropine, dopamine, and isoproterenol on standby.

The patient returned to the OR three weeks later. A temporary transvenous pacemaker was placed prophylactically. Dexmedetomidine and bupivacaine were omitted from the anesthetic. The patient tolerated the procedure well and was discharged home on post-op day 7.

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