Arrest under Anesthesia – what was the culprit?

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Case

- 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 chemo and resection in 2010, portal vein embolization for liver metastases in 2014, and right hepatectomy in 2015, presented to the OR for a hemicolectomy/debulking for tumor recurrence.
- Anesthesia induction was uneventful. Patient received 2mg midazolam, 180mg propofol, 50mg rocuronium, and the airway was secured with 7.5 ETT. Dexmedetomidine infusion was started at 0.4 mcg/kg/hr prior to incision. 20mg of bupivacaine was bolused via the epidural around the time of incision, and epidural infusion of bupivacaine (0.05%) and hydromorphone (8mcg/ml) was initiated shortly thereafter at a rate of 6ml/hr.
- 20 minutes after surgical incision was made, patient acutely developed bradycardia that progressed into asystolic arrest. Code was called, CPR was initiated, and 1mg of epinephrine was administered with return of circulation after 3 minutes.
- Decision was made to abort the procedure and Cardiology was called to evaluate the patient in the PACU.
- Upon detailed evaluation, additional history was obtained of a syncopal event 1.5 years ago, and more recently an episode of lethargy and poor responsiveness a few months ago during which EMT found him to be hypotensive and bradycardic.
- The patient had multiple prior anesthetics without event. However, dexmedetomidine and bupivacaine epidural had not previously been administered.

Discussion

- Bupivacaine toxicity causing arrhythmias and cardiac arrest is well described in the literature. While most theories regarding bradycardia and asystole during spinal and epidural anesthesia involve direct effects of bupivacaine on the heart, inhibition of sympathetic efferents may lead to decreased venous return to the heart which in turn activate 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.(Liguori and Sharrock 1997)
- 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 drop in blood pressure during the infusion. Studies have shown that both sinus and atrioventricular nodal functions can be depressed by dexmedetomidine, and there have been a few case reports describing dexmedetomidine-induced bradycardia culminating in cardiac arrest.(Bharati, Pal et al. 2011)
- A focused yet detailed anesthesia preoperative evaluation can sometimes uncover valuable information that could significantly influence a patient's perioperative management.(Jin and Chung 2001)

Conclusion

- After successful resuscitation, the patient continued to recover uneventfully in the PACU. He was seen by cardiology and transferred to a telemetry floor the next day. Repeat EKG and Echo showed no abnormalities. Troponin was negative as were all other lab values.
- In this case, if history of previous syncope and bradycardia had been documented, the patient would have been referred for a Cardiology consult prior to surgery.
- It was concluded that the patient probably had an undiagnosed underlying sinus node dysfunction. Combined with intraoperative administration of dexmedetomidine and bupivacaine (both associated with bradycardia), these factors likely interacted synergistically to precipitate bradycardia and asystole during surgery. Cardiac Electrophysiology consult recommended preoperative admission and placement of temporary pacing wire prior to rescheduled procedure as well prophylactic atropine, dopamine, and isoproterenol on standby.

References

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