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Case Report

Bradycardia resulting in cardiac arrest in a critically ill patient receiving dexmedetomidine

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ABSTRACT

Dexmedetomidine is an alpha-2 agonist sedative and analgesic used in anesthesia practice, and it has become more prevalent in the critically ill patients requiring short-term mechanical ventilation. While dexmedetomidine is known to have minimal effects on respiratory drive, it has been well-documented to cause bradycardia and hypotension, especially in patients with existing comorbidities. We present a patient without cardiovascular comorbidities who was in the surgical ICU under dexmedetomidine sedation. The patient went into asystole cardiac arrest after vagal stimulation. Return of spontaneous circulation was achieved using ACLS protocol. We offer a review of reported cases and make recommendations on the management of similar situations that may arise given the increasing use of dexmedetomidine.

Introduction

Dexmedetomidine is a selective alpha-2-adrenergic agonist. It is used for sedation, anxiolysis and analgesia. It does not depress the respiratory drive which is a desirable affect in critically ill patients [1]. Moreover, it can be administered in non-intubated patients making it useful in patients who will require short-term mechanical ventilation. While it is known to cause bradycardia and hypotension, little is known regarding the extent of these affects and its interactions with other drugs.

Case report

A 50-year-old female with a past medical history including only alcohol abuse was involved in a motor vehicle collision. She was intubated at the scene for low GCS. On presentation, she was hemodynamically stable. Hydromorphone continuous infusion was initiated on arrival. Imaging revealed bilateral rib fractures, left acetabulum fracture, and intra-abdominal free fluid.

She was taken to the operating room and found to have a full-thickness tear greater than 50% of the bowel lumen in the mid transverse colon with gross spillage. She underwent a colonic resection and left in discontinuity. Dexmedetomidine continuous infusion was initiated on hospital day 0 at a dose of 0.2 μg/kg/h (4.5 mL/h) and titrated to a maximum of 0.6 μg/kg/h (13.5 mL/h on hospital day 2) for ventilator dyssynchrony. She was taken back the following day for abdominal closure and the dexmedetomidine was weaned off. It was restarted at 0300 on hospital day 4 at 0.2 μg/kg/h for ventilator dyssynchrony.

Throughout her hospital stay, she required vasopressor support with norepinephrine (reaching 14 μg/min) and vasopressin thought to be secondary to hypovolemic and distributive shock. Both agents were weaned hospital day 3. Furthermore, given her history of

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alcohol use, she was placed on a phenobarbital taper in addition to thiamine and folate. On hospital day 4, the patient's heart rate dropped about 30 beats per minute (from 97 to 69) after starting the dexmedetomidine at 0.2 μg/kg/h, but her mean arterial pressure remained stable between 65 and 76. Up to this point, daily EKG showed normal sinus rhythm. While having her endotracheal tube suctioned, she went into asystole. ACLS was initiated. She required one round of chest compressions and 1 mg of epinephrine before return of spontaneous circulation (ROSC) was achieved. She remained bradycardic with her heart rate ranging from 30 to 50 despite being on an epinephrine infusion. Cardiology was consulted and performed an echocardiogram demonstrating global hypokinesia with an ejection fraction (EF) of 45% which was a significant change from her echocardiogram three days prior showing an EF 65%. Laboratory values including troponin were unremarkable, and she was later extubated after meeting standard criteria.

Unfortunately, she remained bradycardic and shortly after extubation on dexmedetomidine at 0.2 μg/kg/h, she went into asystole a second time. Atropine was immediately given and ROSC was achieved without chest compressions. Dexmedetomidine was discontinued. Her heart rate improved to 82 beats per minute. She remained asymptomatic and an echocardiogram two weeks later showed an EF back to her baseline of 65%. She recovered without further complications.

Discussion

The popularity of dexmedetomidine has been on the rise following the MIDEX-PRODEX trials which compared midazolam and propofol (respectively) to dexmedetomidine in primarily medical as well as surgical intensive care units. Dexmedetomidine was shown to provide sedation to patients without the effects of respiratory depression, allowing for quicker extubation when compared to midazolam and propofol [2]. This desirable effect propelled its use in surgical intensive care units, especially in patients expected to be extubated within a short amount of time [1,3]. Unfortunately, one of the undesirable side effects of the drug includes its actions on the cardiac system. When compared to midazolam (5.2%), the rate of bradycardia was significantly higher in the group receiving dexmedetomidine (14.2%) [2]. There was also a considerable risk of first-degree atrioventricular block when comparing propofol (0.8%) to dexmedetomidine (3.7%) [2]. Even though these statistics show an increased risk of adverse cardiac affects in patients receiving dexmedetomidine, the rate may be higher if studied in the surgical ICU [2,3].

There have been cases of cardiac arrest reported in patients undergoing neurosurgery with a cardiac history. Bharati et al. concluded that dexmedetomidine should be used with caution in patients who are over the age of 50 or have a cardiac history [4]. The six reported cases occurred within 30 min of drug administration, and five out of the six cases were associated with the administration of a loading dose [4]. A separate series reported three cases of bradycardia with dexmedetomidine and fentanyl, suggesting a synergistic effect [5].

There is conflicting data regarding the extent to which dexmedetomidine effects the cardiovascular system. A pharmacological study showed plasma concentrations up to 0.8 ng/mL were associated with a decrease in catecholamine release and a reduction in mean arterial pressure. Moreover, above 0.8 ng/mL, patients began to exhibit bradycardia and decreased cardiac output [3]. However, other studies have failed to replicate the same dose-dependent bradycardia in the ICU [6].

Whether there is a dose-dependent effect or not, it seems clear that dexmedetomidine is associated with bradycardia in the clinical setting [2]. In addition, it is well established that a vagal response can occur after stimulations such as coughing and gagging and endotracheal intubation to name a few. This triggers a parasympathetic release of acetylcholine to slow the rate at the sinoatrial and atrioventricular node. During such events, bradycardia can be worsened [7,8].

To our knowledge, this is the first case of a patient on dexmedetomidine with bradycardia who went into asystole following vagal stimulation. Though there are many variables that can accentuate the side effects of dexmedetomidine, a trigger like suctioning leading to cough seems to act as a synergistic modifier to worsen bradycardia. We recommend clinicians be cautious with maneuvers that vagally stimulate patients who are bradycardic on dexmedetomidine and have a low threshold for the administration of atropine. Further studies are required to investigate and clarify the effects of dexmedetomidine based on dosage.

Conclusion

Dexmedetomidine has been shown to be a useful drug for sedation in intubated patients. It has minimal effects on the respiratory drive, but it is associated with bradycardia and hypotension. Much is unknown regarding the dose related effects but practitioners should be wary of exacerbating dexmedetomidine-associated bradycardia, especially in the context of vagal maneuvers.

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