Asystole During Administration of Dexmedetomidine with Spinal Anesthesia: A Case Report

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Abstract

Dexmedetomidine is a useful sedative drug that does not cause severe respiratory depression but sometimes causes hypotension or bradycardia. We encountered a case in which asystole occurred during transurethral lithotomy (TUL) that was performed under spinal anesthesia with administration of dexmedetomidine. The patient was a 73-year-old man whose medical history included radiation therapy for prostate cancer and TUL, which had been performed under spinal anesthesia without incident. Results of preoperative examination were unremarkable, and electrocardiography (ECG) showed sinus rhythm with heart rate of 74 bpm. Spinal anesthesia was administered with 3.4 mL of 0.5% hyperbaric bupivacaine, and upper level sensory loss was confirmed at T10. Ten minutes after injection of the bupivacaine, dexmedetomidine was administered for sedation at a loading dose of 3 μg/kg/h over 10 minutes; it was continued at 0.4 μg/kg/h. The patient’s vital signs were stable, but because his SpO₂ on room air decreased to 93%, oxygen inhalation was started, and the dexmedetomidine was reduced to 0.2 μg/kg/h. Upon completion of the operation, 115 minutes after the bupivacaine injection, the patient groaned, and almost simultaneously, his heart rate decreased to 30 bpm and progressed to asystole. The dexmedetomidine infusion was stopped, and 0.5 mg of atropine was injected intravenously. Before chest compressions were started, sinus rhythm returned, and the patient regained consciousness. No ECG abnormalities were found. Upper level sensory block at T10 was reconfirmed. The patient was discharged the next day without complications. We reasoned that the asystole resulted as an adverse effect of the dexmedetomidine, from a vagal reflex, and from the spinal anesthesia. Our case illustrates both the importance of avoiding the administration of dexmedetomidine above the recommended dose during spinal anesthesia and the need for careful ECG monitoring and observation of hemodynamics in patients undergoing TUL.

Key words
Bradycardia, asystole, spinal anesthesia, dexmedetomidine

Introduction

Dexmedetomidine, an alpha-2 agonist, has been used for sedation during spinal anesthesia because it has both sedative and analgesic properties but does not cause severe respiratory depression.¹² In addition, dexmedetomidine has a short half-life, so patients’ return to consciousness is quick. However, dexmedetomidine is well known to cause hypotension and bradycardia. Spinal anesthesia, too, causes bradycardia, due to its sympathetic nerve blocking effect. Although cardiac arrest can occur with the use of dexmedetomidine, such an event is rare. We report the occurrence of severe bradycardia and asystole during urological surgery in patient who was given dexmedetomidine along with spinal anesthesia.

Case report

A 73-year-old man (height, 173 cm, weight, 59 kg) was scheduled to undergo transurethral lithotomy (TUL) for bilateral renal stones. His medical history included radiation therapy for prostate cancer and

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The cardiac rhythm returned to sinus rhythm, and the was normal. The dexmedetomidine infusion was stopped, and 0.5 commands. His heart rate decreased to 30 bpm and the patient groaned, and he failed to respond to verbal placed in each ureter.

Once this was done, the patient’s vital signs remained stable for 1 hour. The TUL was completed 115 minutes after the bupivacaine injection, and a stent was placed in the lithotomy position. Once the spinal anesthesia was administered, his blood pressure was 155/75 mmHg, heart rate was 75 bpm, and SpO₂ was 95%. He was placed in the right decubitus position, and 3 mL of 1% lidocaine was injected for infiltration anesthesia. Dural puncture was performed with a 25-gauge Quincke needle at the L3/L4 interspace via a median approach, and the flow of clear cerebrospinal fluid was confirmed. After 3.4 mL of 0.5% hyperbaric bupivacaine was injected intrathecally, the patient was placed in the supine position. A pinprick test was performed 5 minutes after injection of the local anesthetic, and upper level sensory block was confirmed at T10. At this point, the patient was placed in the lithotomy position. Once the spinal anesthesia took effect, his heart rate decreased to 45–55 bpm, and his blood pressure decreased to 105–120/55–70 mmHg. Ten minutes after the bupivacaine injection, dexmedetomidine was infused for sedation at a loading dose of 3 μg/kg/h over 10 minutes, and this was followed by continuous infusion at 0.4 μg/kg/h. Forty minutes after the dexmedetomidine injection, the patient’s SpO₂ decreased to 93% on room air, so the dosage was decreased to 0.2 μg/kg/h, and oxygen was administered at 4 L/min via face mask. Once this was done, the patient’s vital signs remained stable for 1 hour. The TUL was completed 115 minutes after the bupivacaine injection, and a stent was placed in each ureter.

Immediately after insertion of the ureteral stents, the patient groaned, and he failed to respond to verbal commands. His heart rate decreased to 30 bpm and then progressed to asystole for 32 seconds (Fig. 1). The dexmedetomidine infusion was stopped, and 0.5 mg of atropine was injected intravenously. Before we initiated mask ventilation and chest compressions, the cardiac rhythm returned to sinus rhythm, and the patient regained consciousness. The ECG recording was normal.

Cold testing at the end of the surgery confirmed upper level sensory block at T10. The surgery had lasted 100 minutes, and the anesthesia had lasted 130 minutes. The intraoperative fluid volume was 390 mL, and the intraoperative blood loss volume was <10 mL. Upon leaving the operating room, the patient was monitored for 24 hours. There were no clinical events, and he was discharged on postoperative day 1 without neurological or cardiac dysfunction. Two years have passed since the surgery, and the patient has had no complaints.

Discussion

We assume that the asystole in this case was the result of the combined effect of dexmedetomidine, the spinal anesthesia, and a vagal reflex.

Dexmedetomidine has been shown to decrease heart rate. Bradycardia is reported to occur in 9–42% of mechanically ventilated intensive care unit patients given dexmedetomidine.1–13 Dexmedetomidine, an alpha 2 agonist, is a useful sedative drug for local anesthesia and for neuraxial anesthesia, especially because it has opioid-sparing and sympatholytic effects, and as an analgesic, it has a long-lasting effect without producing severe respiratory depression.4–11 Lee et al reported that bolus administration of 0.5 μg/kg or 1.0 μg/kg dexmedetomidine prolonged the duration of spinal anesthesia with 0.5% bupivacaine (time to motor block regression: control vs. dexmedetomidine, 98.8 ± 34.1 vs. 132.9 ± 43.4 minutes, respectively; P<0.05).5–8 Dexmedetomidine provides high-quality sedation and generates natural sleep,6 and thus, its use has spread worldwide. However, there have been several reports of cardiac arrest with the use of dexmedetomidine.7–8 Bharati et al warned that indiscriminate use of dexmedetomidine could increase the incidence of cardiac arrest occurring during general anesthesia, especially, in patients who are older than 50 years of age and those who have an underlying cardiac disorder or when used in combination with a cardiodepressant drug.9 The bradycardia that accompanies the use of dexmedetomidine is thought to be the result of decreased central sympathetic output, decreased catecholamine release, and increased central parasympathetic output. Sharp et al, using a whole-cell voltage clamp methodology, showed that dexmedetomidine decreased both GABAergic and glycinergic inhibitory input to cardiac vagal neurons but had no significant effect on excitatory input in the rat nucleus ambiguus.10 This means that decreasing inhibitory neurotransmission to cardiac vagal neurons increases the excitability of parasympathetic neurons, leading in turn to bradycardia.
Figure 1. Lead II electrocardiography recordings (paper speed 25 mm/sec).

a: 14:00--Before spinal anesthesia (after patient’s admission to the operating room). Heart rate was 76 bpm. 
b: 14:06--After spinal anesthesia. Heart rate was 52 bpm. 
c: 16:01:50–16:02:10--After insertion of the ureteral stents, heart rate dropped suddenly to 30 – 40 bpm. 
d: 16:02:20 – 16:02:40--Bradycardia and sinus arrest. 
e: 16:02:50 – 16:03:20--Sinus arrest continued for 32 seconds. 
f: 16:03:30--Heart rate returned to sinus rhythm; heart rate was 55 bpm.
Thus, atropine, as an anticholinergic drug, is effective for the bradycardia induced by dexmedetomidine. Administration of atropine was effective in restoring sinus rhythm in our patient, an outcome that supports the hypothesis described above.

Whether atropine can be used as premedication to prevent bradycardia during spinal anesthesia is controversial. Ahn et al reported that intravenous injection of atropine significantly reduced the incidence of bradycardia during spinal anesthesia.11) Hirabayashi et al, however, reported that intramuscular injection of atropine had no influence on the incidence of bradycardia occurring during spinal anesthesia.12)

There has been only a single reported case of cardiac arrest after the administration of dexmedetomidine with spinal anesthesia. Kim et al reported the occurrence of severe bradycardia within 12 minutes after dexmedetomidine injection that was followed by asystole within 10 seconds later. Atropine and epi-nephrine injection were used for cardiopulmonary resuscitation in this patient, and she recovered without any complications.8) To the best of our knowledge, our report is the second report of cardiac arrest after injection of dexmedetomidine with spinal anesthesia. In comparison to the patient reported by Kim et al, our patient had no preoperative ECG abnormalities. Furthermore, our patient’s bradycardia and asystole did not occur as quickly as they did in the patient reported by Kim et al. This means that the dexmedetomidine alone may not have been fully responsible for the change in our patients’ cardiac rhythm.

Both hypotension and bradycardia are common during spinal anesthesia. Hypotension is reported to occur in up to 22% of patients during spinal anesthesia, and bradycardia is reported to occur in as many as 10%.13) In their review, Kinsella and Tuckey noted that the main factor implicated in these complications was dermalomal block to T5/6 or higher. The decrease in heart rate is a result of blockade of cardioaccelerator fibers arising from T1-T4.14)

High spinal anesthesia with T1-level blockade or higher, i.e., blockade of cardioaccelerator fibers, in addition to marked reduction in venous return has been shown to result in severe bradycardia and even asystole because of unopposed parasympathetic activity.15) However, Carpenter et al found no correlation between the level of sensory blockade and the magnitude of bradycardia.13) In our case, sensory blockade at T10 was confirmed immediately after surgery, and the patient’s heart rate before the adverse event was steady (45–55 bpm). Thus, we surmise that blockade of sympathetic nerves during spinal anesthesia was not a strong influence on the severe bradycardia and asystole in our patient.

A cardiac vagal reflex is another possible cause of severe bradycardia and asystole during spinal anesthesia. Løvsrad et al reported five cases of asystole or bradycardia during spinal anesthesia. They suggested cardiac vagal reflex induced asystole.15) The Bezold-Jarish reflex in particular may occur in the presence of hypovolemia, with the small volume triggering mechanoreceptor-mediated bradycardia.16) Positional change during surgery may also trigger a cardiac reflex.15)

Guan and Liu described 2 interesting cases of hypotension and bradycardia that occurred during transurethral resection of the prostate.17) They concluded that a parasympathetic reflex caused severe hypotension and bradycardia. They also assumed that a prostate procedure may induce a vagal reflex because the pelvic splanchnic nerves, which are parasympathetic nerves, run close to the apex of the prostate at the level of the membranous urethra.17) The prostate was not manipulated directly in this patient, lower urinary tract manipulation with endoscopic instruments during ureteral stents placement might have affected pelvic splanchnic nerves around the prostate. So, the transurethral procedure might have increased parasympathetic activity and induced a cardiac vagal reflex. However, there were no reports of sinus arrest or cardiac arrest induced by a vagal reflex during ureteral stent procedure.

Our patient had not been taking any medication, such as a beta-blocker, that could have affected his heart rate, nor was a rhythm abnormality found upon preoperative electrocardiography. When the patient was given bupivacaine intrathecally, his heart rate decreased to 45–55 bpm, but such slowing is commonly seen with spinal anesthesia.

The details of our case, along with findings reported in the literature, have led us to conclude that a cardiac vagal reflex caused our patient’s severe bradycardia and asystole. We also speculate that blockade of sympathetic nerves resulting from the dexmedetomidine and the spinal anesthesia led to a decrease in the basal heart rate and induced the cardiac vagal reflex.

We conclude that our case illustrates, at the very least, both the importance of avoiding administration of dexmedetomidine above the recommended dose during spinal anesthesia and the need for careful ECG monitoring and observation of hemodynamics.
Asystole under dexmedetomidine sedation

in patients undergoing TUL.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report.

References