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CASE REPORT

Mobitz type II second-degree atrioventricular block after intravenous infusion of dexmedetomidine in an elderly patient undergoing radical prostatectomy: a case report

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ABSTRACT

Dexmedetomidine is an α2-adrenoceptor agonist widely used in anesthesiology. However, its indiscriminate use may result in significant adverse effects. We report a case of dexmedetomidine-induced Mobitz type-II second-degree atrioventricular block in an elderly hypertensive patient undergoing radical prostatectomy. After receiving dexmedetomidine at a dose of 1mcg/kg, the patient exhibited a pronounced bradycardia (heart rate 30-40 bpm) with unsatisfactory response to anticholinergics. Urgent cardiology evaluations confirmed the Mobitz type-II second-degree block diagnosis, and the surgical procedure was postponed for further assessment. The patient remained stable without the need for the implantation of a temporary pacemaker. This case highlights the potential complications associated with the use of dexmedetomidine in elderly patients with pre-existing cardiac conditions.

KEYWORDS

Dexmedetomidine; bradycardia; intubation; atrioventricular block

INTRODUCTION

Dexmedetomidine is a widely used adjuvant agent for intravenous anesthesia in anesthesiology and intensive care due to its analgesic, anti-inflammatory and antihyperalgesic properties[1]. However, improper use of this selective α2-adrenoceptor agonist may be associated with significant complications[1,2]. One of the most prevalent adverse effects associated with dexmedetomidine is bradycardia. This is a consequence of the drug's agonistic action on presynaptic and imidazoline α2-receptors, which ultimately results in unfavorable antiadrenergic and chronotropic effects[1].

A number of publications have highlighted safety concerns associated with the improper use of dexmedetomidine, emphasizing the risks of severe bradycardia and cardiac arrest[2]. In light of this, we report a case of an elderly, hypertensive male patient with a first-degree atrioventricular (AV) block who subsequently developed a Mobitz type-II second-degree AV block after dexmedetomidine administration. This case report provides further insight into the potential cardiovascular effects of dexmedetomidine and highlights the importance of careful patient selection and monitoring when using this drug in clinical practice.

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CASE REPORT

The patient provided informed consent, and this case report was approved by the local ethics committee (CAAE-67385823.0.0000.0257). This case report was described following the CARE report guidelines[3].

A 72-year-old, 170 cm (5.61 feet), 70 kg (154.3 pounds) male was admitted for a laparoscopic radical prostatectomy. The patient had a past medical history of chronic bladder retention due to prostate adenocarcinoma and had been using an indwelling bladder catheter for a period exceeding 18 months.

The patient presented with a documented history of arterial hypertension, pre-diabetes (defined as fasting plasma glucose levels between 100-126 mg/dL), and dyslipidemia, along with a history of acute myocardial infarction in 2001 and transient ischemic attack in 2018. The patient's medication regimen included losartan (100mg/day), amlodipine (10mg/day), metformin (500mg/day), acetylsalicylic acid (100mg/day), clopidogrel (75mg/day), simvastatin (40mg/day), and omeprazole (20mg/day).

The patient's clinical history indicated a functional capacity exceeding 4 metabolic equivalents (MET). Preoperative complementary tests revealed the patient had moderate renal dysfunction, with a creatinine clearance of 43ml/min as assessed via the Cockcroft-Gault equation. His physical examination showed a Mallampati III score, reduced thyromental distance and cervical mobility. Additionally, an electrocardiogram showed the presence of first-degree AV block, and ambulatory blood pressure monitoring revealed nocturnal elevations in both systolic and diastolic blood pressures, with the highest value recorded at 199/123mmHg during sleep. In addition, the patient exhibited no clinical signs or symptoms indicative of obstructive sleep apnea syndrome. Given these findings, the recommended anesthesia strategy involved the use of continuous epidural anesthesia in combination with balanced general anesthesia.

Upon arrival in the operating room, the patient was evaluated and deemed to be in satisfactory general condition, with an intact orientation and no complaints. A 20G venous catheter was inserted into the right median cubital vein, and multiparametric monitoring was initiated, including electrocardiography monitoring, pulse oximetry, and non-invasive blood pressure (NIBP) measurements. No significant alterations were observed in the monitored vital signs. The cardiac rhythm was regular, with the presence of first-degree AV block and a heart rate (HR) of 85 bpm.

To induce anxiolysis for epidural anesthesia, the patient was given 2mg of intravenous midazolam, 50mcg of fentanyl as a bolus, and 70mcg of dexmedetomidine (1 mcg/kg) infused over 10 minutes. While the HR decreased to 60 bpm, all other vital signs remained within the normal range. Next, an epidural puncture (aseptic technique) was performed. We used a 17G Tuohy needle at spaces L3-L4, identified via the loss of resistance approach to saline. The epidural catheter was inserted without complications, and 5 mL (100mg) of 2% lidocaine with epinephrine was administered.

The anesthetic depth was monitored using the bispectral index (BIS®). The patient was positioned and pre-oxygenated with a high-flow oxygen face mask for induction of general anesthesia. Subsequently, 150mcg of fentanyl, 50mg of propofol, and 10mg of cisatracurium were administered. Despite a Cormack IIIA view during direct laryngoscopy, significant bradycardia was observed on the monitor, with values oscillating between 30-40 bpm (Figure 1 and **video 1** https://drive.google.com/file/d/14XvSXnVTfQ6SeOUYDfNoYY0ilDBvph5b/view?usp=sharing).

An additional two intubation attempts were made by a second anesthesiologist, preceded by a venous bolus of atropine (0.5 mg and 1 mg). This resulted in a slight increase in heart rate to 55-60 bpm. However, there was a second significant drop in heart rate during laryngoscopy (averaging 30-40 bpm). Blind intubation was completed on the third attempt with the use of an 8.0 orotracheal tube with a cuff, confirmed by the capnography curve. Following intubation, the patient was placed on mechanical ventilation and exhibited stable hemodynamic parameters. This was evidenced by a non-invasive blood pressure of 102/65 mmHg, peripheral oximetry of 98%, and capnography registering an end-tidal CO2 level of 38 mmHg. However, the patient remained persistently bradycardic, with an HR of 30-40 bpm. A subsequent bolus administration of adrenaline (5 mcg) resulted in an inadequate response (Figure 2).

An urgent cardiology assessment was requested. A 12-lead electrocardiogram confirmed the diagnosis of second-degree Mobitz II AV block. The cardiology team recommended postponing the surgery to conduct additional diagnostic evaluations. The patient remained hemodynamically stable and the implantation of a temporary pacemaker was not required.

Neuromuscular block reversal was achieved using 1 mg of atropine and 2.5 mg of neostigmine. The patient was successfully extubated, and the epidural catheter was removed without any complications. The patient was subsequently transferred to the post-anesthesia care unit in a satisfactory general condition, though exhibiting a degree of somnolence. At the time of transfer, the patient's ECG showed a second-degree Mobitz II AV block with an HR of 30-40 bpm, an NIBP of 128/70mmHg, and SpO2 at 96% while receiving oxygen via a nasal catheter (2 litres per minute).

Figure 1. Electrocardiogram tracing showing second blocked P wave (yellow arrow) (second-degree atrioventricular block type Motiz II).

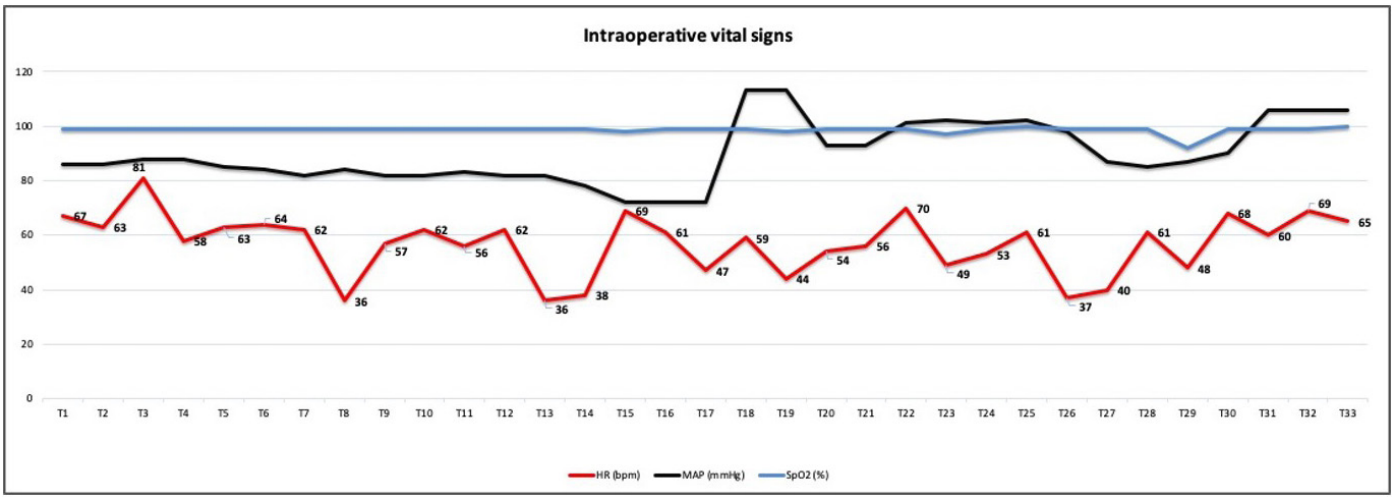

Figure 2. The values were recorded on the multiparametric monitor used during the procedure, and grouped into 33 different time intervals (T1-T33). After the patients' admission, the first recording was identified as T1 and occurred at 8:56 and the last measurement was at 10:00 (T33), with an interval of 2-5 minutes between each measurement. Significant bradycardia was identified primarily during the procedure (T8), evolving with significant variability depending on the treatment used. However, with no repercussions on MAP. Only after the administration of adrenaline as a second-line agent for treating complications (T18-T20) were pressure changes observed; however, these changes were not found to have any lasting efficacy in maintaining a heartbeat. HR: heart rate; MAP: mean arterial pressure (mmHg); SpO_2: Peripheral oxy-hemoglobin saturation.

DISCUSSION

In this clinical report, we presented an atypical case of an elderly male patient with multiple risk factors for intraoperative instability who developed a Mobitz type-II second-degree AV block following the administration of dexmedetomidine. The patient experienced refractory bradycardia (with a heart rate of approximately 38 bpm) during and after challenging intubation procedure. The procedure was rendered unfeasible when a transition from first-degree to second-degree AV block was identified.

Over the past decades, international guidelines have been developed, and various pharmacological approaches have been recommended to prevent anesthesia-related complications[4,5]. These preventive measures include using local anesthetics, beta-blockers, and α2 agonists during the induction of anesthesia[1,5,6].

In this respect, dexmedetomidine, a pre-synaptic α2-adrenoceptor agonist, is believed to be a protective agent against tracheal intubation-related complications by providing antinociceptive effects, stabilizing ion channels and acting as an antagonist to the adrenergic system[1]. Moreover, α2 agonists affect the dorsal horn and brainstem nuclei, resulting in sedative, analgesic, and antihyperalgesic effects[1,7]. Given the advantages of dexmedetomidine during tracheal intubation, its

extensive utilization may occur without sufficient attention to the potential risks.

The development of second-degree AV block during intubation has been hypothesized to be related to multifactorial causes, including the fragility of the cardiac conduction system due to aging, synergism, and dosage of drugs, as well as the the frequency and duration of intubation attempts. For example, Nagasaka et al.[8] reported a case of an elderly female patient with arterial hypertension, type-II diabetes and asymptomatic first-degree AV block. The patient underwent rectal resection and received dexmedetomidine, resulting in a second-degree AV block. The condition subsequently progressed to a complete AV block and cardiac arrest[8].

Peden et al.[9] conducted a randomized trial to evaluate the benefits of dexmedetomidine in 49 patients who were administered alfentanil and propofol for anesthesia induction. The authors found that two patients who received higher doses of dexmedetomidine experienced cardiac arrest, requiring a dose reduction.

Bharati et al.[2] presented a case series comprising six patients who had received dexmedetomidine and subsequently experienced cardiac arrest. This finding suggests a correlation between advanced age and pre-existing cardiovascular comorbidities. Specifically, five patients were over 50 years old, and all exhibited cardiovascular comorbidities before receiving dexmedetomidine. According to the authors, four patients who experienced cardiac arrest received the loading dose prior to induction[2].

As reported in the case series by Bharati et al.[2], our patient received propofol and fentanyl for anesthesia induction, which may have potentiated the bradycardic action of dexmedetomidine. The same pattern can be identified in the study by Peden et al.[9], in which patients who received propofol and alfentanil also experienced complications. Thus, different drugs' synergism (e.g., additive effects) must always be considered. It is crucial to adjust the dosage of dexmedetomidine when used in conjunction with other harmful inotropic and chronotropic agents, particularly in older patients and those with cardiac conditions. Etomidate could be considered an alternative to propofol[10].

Furthermore, we cannot rule out that laryngoscopy in susceptible patients may trigger or potentiate the deterioration of the conduction system. The primary manifestations of laryngoscopy in adult patients are typically hypertension and tachycardia, which is why dexmedetomidine was indicated for systemic protection. Bradycardia is rarely reported and may be related to conduction system disorders such as first-degree AV block which is linked to vagal stimulation[11]. Therefore, adrenergic antagonist agents in this population should be used with caution. In this regard, two meta-analyses evaluated the effects of perioperative dexmedetomidine. Li et al.[12] observed no differences in the incidence of bradycardia or hypotension in patients undergoing cardiac surgery. In contrast, Zeng et al.[13] observed a reduction in the incidence of hypertension and tachycardia and an increase in the incidence of bradycardia in elderly patients undergoing noncardiac surgery without an increase in the incidence of hypotension. Despite this, the risk of bradycardia and hypotension cannot be ruled out in this population. It may be advisable to consider a reduced dose of dexmedetomidine, as well as delaying or even avoiding the bolus dose.

In summary, we report a case of Mobitz type-II second-degree AV block after the administration of dexmedetomidine during tracheal intubation in an elderly male patient with cardiovascular history. Although dexmedetomidine is frequently regarded as a safe and efficacious agent, there is an opportunity for further research and discussion concerning the potential association between this selective α2-adrenergic agonist and cardiac conduction system abnormalities. Additional studies could provide valuable insights that may ultimately enhance patient outcomes. For high-risk patients with pre-existing cardiac conduction system abnormalities, we conclude that caution is essential when monitoring and adjusting the dosage of dexmedetomidine to mitigate the risk of serious adverse events.

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