Case report: severe bradycardia following spinal anesthesia

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Abstract

Introduction: The prevalence of severe and life-threatening bradycardia following spinal anesthesia, mostly associated with high spinal block, is about 2.5 percent. Numbness levels higher than T4 are considered a risk factor for occurrence of severe bradycardia and even cardiac arrest. This study presents an 81-year-old male patient presenting to Peymanieh Hospital in Jahrom to undergo a surgery for inguinal hernia and hydrocele. Until 13 minutes after spinal blocking, all vital signs were normal, but without symptoms of high spinal blocking, the patient's heart rate suddenly dropped to about 45 bpm, and a little later to 22 bpm. Fortunately, heart rate returned to normal with injection of atropine and ephedrine.

Conclusion: The incidence of severe and life-threatening bradycardia is not necessarily associated with high spinal blocking and can occur even in the presence of normal spinal block. This fact highlights the importance of careful and real-time patient monitoring.

Keywords: Spinal Anesthesia, Bradycardia, Inguinal Hernia, Hydrocele

Introduction

This case study introduces an 81-year-old man (67 kg, 165 cm) without a history of a serious disease or medication. He had undergone a surgery for inguinal hernia and hydrocele in Peimanieh Hospital in Jahrom, Iran. He had a history of prostatectomy that had caused no complications. The patient's electrocardiogram in the pre-surgical anesthesia consultation showed a normal sinus rhythm and no signs of ischemia, hypertrophy, and heart blocks (Electrocardiogram 1).

The pre-surgical tests, including measurement of potassium (4.4 mmol), sodium (144 mmol), fasting blood sugar (78 mg/dl), platelets (445000 µl), hemoglobin (12 g/dl), heart beats (62 bpm), systolic blood pressure (115 mmHg), and diastolic blood pressure (80 mmHg) were all normal.

The patients’ hemodynamic conditions while entering the operation room was completely stable and appropriate (heart beats of 76 bpm, systolic blood pressure of 126 mmHg, & diastolic blood pressure of 74 mmHg). The patient received fluid...
therapy while entering the operation room and underwent spinal anesthesia under standard conditions. Once the puncture site (L4-L5 intervertebral space) was determined, the site was disinfected with betadine and then dried. A needle for spinal anesthesia (gauge 25, orange, Quincke) was then entered into the subarachnoid space through the intervertebral space, and 3 cc (15 mg) of 0.5% marcaine was injected after observing cerebrospinal fluid at the needle hub. The surgery was eventually performed with the patient in the prone position after ensuring the appropriate level of anesthesia and sterilizing the site of surgery. All vital signs of the patients were normal and acceptable for the first 13 minutes after the spinal puncture, but his heartbeat suddenly decreased to 45 bpm before incision. He became a little drowsy but still remained conscious although his consciousness had decreased. Given the situation, 0.6 mg of atropine was injected intravenously, but his heartbeat decreased to 22 bpm, and thus, 10 mg of ephedrine was injected. Ten milligrams (10 mg) of metoclopramide was injected due to his nausea. His heartbeat increased to the normal level (86 bpm) 45 seconds after injecting ephedrine. His blood pressure also reached 145.88 mmHg. The patient's level of anesthesia was examined to ensure the lack of high anesthesia, and no high level of anesthesia was observed (anesthesia at T10). About ten minutes after the patient's status normalized, and appropriate stable hemodynamic conditions were ensured, the surgery team was allowed to begin the surgery, and no special problem occurred until the end of the surgery. The patient's conditions were favorable during one hour in the recovery room. He was transferred to the cardiac care unit after the surgery to be further sure about his safety. No significant changes were observed between the electrocardiogram before surgery and after surgery (Electrocardiogram 2). The patient was discharged from the hospital the next day in a good general condition. The patient and his family were suggested to see the cardiologist seven days later for a reassessment.

Electrocardiogram 1
Discussions
Severe and life-threatening bradycardia following spinal anesthesia occurs by 2.5% (1) and is often accompanied with a relatively high level of anesthesia (2). The anesthesia higher than T4 is known as a risk factor for severe bradycardia and even cardiac arrest. The reason is that bradycardia occurs when T1-T4 nerve fibers responsible for increasing heartbeat are blocked (1). Those nerve fibers are called cardioaccelerator. Cardiac arrest has also been reported following spinal anesthesia in caesarean section when placental is being pulled out and during uterus massage for controlling hemorrhage (3). Pulling and manipulating the visceral peritoneum and ligaments during abdominal surgeries cause severe bradycardia and hypotension (4). Bradycardia happening in surgeries following spinal anesthesia is usually caused by suppression of a cardiovascular reflex known as Bezold–Jarisch reflex that reduces venous return to the heart and thus reduces cardiac output (5). The standard treatment in that situation is the quick and timely injection of atropine (6). Maitra et al. reported a case of a 31-year-old woman who had been presented for a caesarean section under spinal anesthesia. Fifteen (15) minutes after induction of anesthesia at T8 level when the child was born and her uterus was being massaged, severe bradycardia with almost 29 bpm and nausea occurred without any signs of high anesthesia. The heartbeat normalized after injecting atropine, and the surgery was continued. Her heartbeat again decreased from 90 bpm to 65 bpm and then increased while the uterus was being massaged. Yong Hong Chung et al.’s study (8) presented a 39-year old woman who was at gestational age of 37 weeks and was admitted to the maternity ward for induction of anesthesia because of her preeclampsia and intrauterine growth retardation. She received oxytocin infusion and spinal anesthesia. After two minutes of being in left recumbent position, she was spinally anesthetized with 20 mg of fentanyl and 3.6 mg of ropivacaine, the embryo’s heartbeat decreased to 50-70 bpm. The mother’s blood pressure was 135.70 mmHg at that time. Regarding the situation, oxytocin infusion was stopped immediately, and she underwent fluid therapy in the same position. As the embryo’s heartbeat did not increase, an emergency cesarean section was performed, and the infant with an Apgar score of 5 was born and transferred to the neonatal intensive care unit after resuscitation and intubation. Hemorrhage was observed in the right hemisphere in infant’s brain CT scan.

The significant point of the mentioned case study is the occurrence of severe life-threatening bradycardia without signs of high anesthesia in the patient. Although moderate bradycardia occurring after spinal anesthesia is usually due to the sympathectomy caused by anesthesia, severe bradycardia occurs at high levels of anesthesia when T1-T4 nerve fibers responsible for increasing heartbeat are
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 blocked. However, the patient discussed in this case study did not show any signs of high level anesthesia, which differentiates this case from other cases of prevalent bradycardia occurring after spinal anesthesia.

**Recommendation**

It is recommended to attend to the cardiovascular monitoring, especially the electrocardiogram monitoring, arterial oxygen saturation, and heartbeat before induction of spinal anesthesia.

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**Conflict of Interest**

The authors declare no conflicts of interest regarding the compilation/publication of this article.

**References:**
