

# Sudden syncopal attack after postobstructed diuresis under combined spinal epidural anesthesia

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Micturition is clinically well-known as a factor that may cause hypotension, bradycardia, and if severe, syncope. However, the mechanism of syncope during micturition is not completely understood. Vasodilatation, bradycardia, and reflex cardiovascular depression are all called by various terms including vasovagal syncope, Bazold-Jarisch reflex, and neurocardiogenic syncope [1]. The neurally-mediated syncope includes true and situational syncope that can take place in various situations such as blood loss, cough, sneeze, micturition and lifting up a heavy load.

A case of a 71 year-old man under combined spinal epidural anesthesia for total knee replacement, in which the operation were performed without knowing that a Foley catheter installed had been obstructed, and severe bradycardia, hypotension, and loss of consciousness occurred immediately after moving the patient to the recovery room following micturition were described.

The patient had a history of syncope. Preoperative cardiac test was normal. At the operation room, heart rate and blood pressure were 70 /min and 170/90 mmHg. Beginning with the sufficient administration of fluid, the regional anesthesia was performed and the sensory block was verified up to the T4 dermatome. A forced air warmer was used to maintain the body temperature. At the end of operation, 8 ml of 0.15% levobupivacaine was injected through the epidural catheter, and then a mixture of anesthetics was continuously injected. Intraoperative systolic blood pressure were ranged from 110 to 190 mmHg, diastolic pressure 65 to 105 mmHg, heart rate 52 to 70 /min, and estimated blood loss was approximately 500 ml. Although the total amount of fluid was approximately 1,000 ml, no urine out-

put was observed. Just before leaving the operation room, it was found that the tip of the Foley catheter was obstructed. When the catheter was opened, about 700 ml of urine was discharged. At that time, the vital signs were normal and consciousness clear. Finding that the sensory block level had come down to the T12 dermatome, the patient was moved to the recovery room. Immediately after arriving at the recovery room, the heart rate was drastically reduced from 70 /min to 30 /min or below, the blood pressure was 50/30 mmHg, and consciousness was lost. Promptly, 0.5 mg of atropine sulfate and 10 mg of ephedrine were administered, and the patient-controlled epidural analgesia (PCEA) was stopped. Following the treatment, the heart rate recovered to 60 /min and the blood pressure to 110/80 mmHg, and subsequently, the consciousness of the patient was recovered. While the patient remained in the recovery room, no abnormal findings were observed. Micturition syncope was confirmed that head-tilt test done after the operation was positive.

The main causes of consciousness loss when awakening from anesthesia include metabolic anomalies such as hypoglycemia, hyponatremia, hypoxia and severe cardiovascular collapse. However, based on the fact that the patient had no findings of cardiac abnormalities, the patient in this case might have experienced reflex micturition syncope. Micturition syncope is one type of situational syncope that occurs due to neural mediation, and usually occurs in healthy men after alcohol hyperingestion [2]. Sumiyoshi et al. [2] mentioned sleeping under warm blankets and administration of a vasodilator can be factors that cause a reduction in the peripheral arterial blood resistance in the lower extremities and reported that the Valsalva maneuver is a factor

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that causes neurally-mediated reflex action after micturition. In our case, the sensory block to the T4 dermatome might have vasodilator effect, and warm blanket might have caused mild vasodilatation. The blood volume might have not been sufficient, considering that duration of the operation was about three hours.

Hainsworth [3] mentioned emotional stress, Valsalva maneuver, and hypovolemia as induction factors which caused syncope. In our case, when the urinary bladder was full, abdominal pressure might have been increased with all the intraperitoneal blood vessels. In the blood vessels outside the abdominal cavity, venous return might have been limited by the full urinary bladder and cardiac output gradually reduced. Moreover, it is assumed that opening the blocked Foley catheter caused blood-stream inhibition of the venous return by a loss in the stretch receptor stimulation. In summary, taking into consideration the warm blanket, vasodilatation by sensory block, hypovolemia, the PCEA initial dose, Valsalva maneuver, and a history of syn-

cope, it is assumed that micturition syncope caused the severe bradycardia, hypotension, and loss of consciousness. The diagnosis of micturition syncope is done by a head-tilt test in which the bed is tilted by 60–70° in parallel with an investigation of the patient's history, physical examination, and electrocardiogram. It was confirmed that head-tilt test after the operation was positive. The specificity of the tilt test with normal volunteers and with patients who have a history of typical neurally-mediated syncope, was reported about 90% and sensitivity ranges from 32 to 85%, respectively [4].

In conclusion, when performing combined spinal epidural anesthesia in a patient who has a history of syncope and is supposed to undergo lower extremity surgery, a sufficient volume of fluid should be supplied, and factors that may reduce venous return should be eliminated. If necessary, in a patient in whom syncope is anticipated, it would be helpful to perform head-up tilt test before the operation. Moreover, it should not be forgotten to check whether the Foley catheter is obstructed or not.

## References

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