Letter to the Editor

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Accidental intrathecal injection of dopamine hydrochloride resulting in analgesic effects

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Medication errors are defined as any error in the delivery process, whether there are any adverse consequences or not [1]. In the present case report, we describe an accidental intrathecal injection of dopamine hydrochloride (DA) which has not been previously reported in human. We were blinded to the fact due to the analgesic and anesthetic effects that seemed to have been achieved after the injection of DA.

A 76 year-old man was scheduled for closed reduction and internal fixation of an intertrochanteric fracture of the right femur. His medical history included diabetes mellitus, hypertension and chronic renal failure with maintenance hemodialysis for the last 4 years. The blood glucose level was with 95–337 mg/dl and the blood pressure (BP) with 140–200/80–100 mmHg poorly controlled. The preoperative pulmonary function test revealed a severe restrictive respiratory disorder. We decided to apply a spinal anesthesia for his operation.

Electrocardiogram, BP, heart rate (HR) and arterial hemoglobin oxygen saturation were measured after the patient was brought to the operating room. The BP was 160/80 mmHg and the HR showed 95 beats/min. A spinal anesthesia was performed at the L3-4 interspace with the patient in a left lateral position using a 25-gauge Quinke tip needle. The drug ampule assumed to content of 0.5% bupivacaine hydrochloride (20 mg/4 ml, Hana Pharm, Seoul, Korea) was passed by the nurse and 2.0 ml of drug was injected into the intrathecal space. Five minutes later, he was not able to sense cold sensation (alcohol swab) and pain (blunt needle) below T10 level and the surgery began.

After skin incision, the patient seemed comfortable and the vital signs remained constant except a slight increase in BP with around 170/90 mmHg. In 30 minutes after incision, he complained of mild pain at the incision site. Shortly after, the nurse noticed that 80 mg of DA (200 mg/5 ml, Hana Pharm, Seoul, Korea) had been drawn and intrathecally injected instead of 0.5% bupivacaine (Fig. 1). 80 mg propofol was intravenously administered and a laryngeal mask airway (LMA) was inserted. Anesthesia was maintained using 0.5–1.0 MAC sevoflurane. We performed an arterial cannulation at the left radial artery and continuously monitored the arterial pressure. After induction

Fig. 1. Ampules of bupivacaine (left) and dopamine hydrochloride (right). They are similar in size, color, and shape.

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of the general anesthesia, the BP decreased to 90/60 mmHg and the HR were decreased to 90 beats/min but the intraoperative course was uneventful. The surgery lasted for 1 hour and the LMA was removed thereafter. The patient was transferred to the intensive care unit (ICU) for observation. The patient was alert and BP and HR were 160–190/70–80 mmHg and 90–110 beats/min respectively. He complained of mild pain (VAS 3) soon after arrival at the ICU. Thirty minutes later, he complained of severe pain (VAS 7) which required additional analgesia. His pain was subsided after intravenous injection of 100 ug fentanyl. The motor and sensory functions that were examined by a neurologist at the ICU showed unremarkable findings. Twenty four hours later, the patient was transferred to the general ward. After two weeks of observation, he was discharged without any adverse events. In spite of our recommendation, he did not revisit the hospital within 1 month after discharge. We called him instead at 1 and 6 months after discharge. He was able to walk again and there were no remarkable complications.

The present case illustrates a medication error whereby a wrong medication was administered to the intrathecal space. DA is easily available in the operating room as intravenous medication acting on the adrenergic and dopaminergic receptors, producing effects such as increased BP and HR. The intrathecal injection of DA has never been previously reported in humans. However, intrathecal dopaminergic agents have been used in studies of antinociceptive properties of spinal dopamine receptors in animals [2-4]. Increasing evidence suggests that the central dopamine system is involved in the modulation of nociception at the supraspinal and the spinal cord levels. Focal electrical stimulation in the origin of the A11 area suppresses the nociceptive responses of neurons in the spinal dorsal horn [2]. Yang et al. [3] demonstrated that intrathecal administered DA (up to 16.5 nM) produced significant and dose-dependent prolongation of the tail-flick latency. Recently, a higher dose of DA (100 uM) was injected in the spinal cord of rats demonstrating dopaminergic antinociceptive actions [4]. Besides dopaminergic receptors, it has been well known that α2-adrenergic receptors in the spinal cord produce a dose-dependent antinociceptive effect [5]. When a large dose of DA is administered intravenously, α-adrenergic activation is dominant. However, it is not known whether this dose-dependent receptor affinity of intravenous DA can be applied equally to intrathecally administered DA.

It is difficult to suggest the clinical use of DA as an intrathecal analgesic from this case report, because there are no evidences to prove any advantages of DA compared to local anesthetics or α2 agonists. Moreover, although there was no clinical neurologic complication in this case, DA can induce vasoconstriction in the spinal cord, which might result in ischemia and irreversible spinal cord injury. However, numerous studies have demonstrated that spinal dopaminergic and adrenergic receptors have shown to play critical roles in pain modulation. Since DA stimulates both receptors and our case report showed an analgesic effect of intrathecal DA, it intrigues us to find a contributing role of intrathecal DA in the analgesic management.

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