Multiple system atrophy (MSA) is a progressive neurodegenerative disorder characterized by extra-pyramidal symptoms, cerebellar ataxia, and autonomic dysfunction. The perioperative management of patients with MSA is challenging primarily due to autonomic dysfunction and vocal cord paralysis. Dexmedetomidine (DEX) is a highly selective $\alpha_2$-adrenoreceptor agonist that is used as a sedative, causing minimal depression of the respiratory function. We administered DEX continuously for sedation during spinal anesthesia for bipolar hip arthroplasty in a patient with MSA, without any cardiovascular or respiratory complications. This is the first case report of the efficacy of DEX for the sedation of a patient with MSA during spinal anesthesia.

**Key words:** Multiple system atrophy, Dexmedetomidine, Spinal anesthesia

**INTRODUCTION**

The perioperative management of patients with MSA, a progressive neurodegenerative disorder, is challenging, primarily due to autonomic dysfunction and vocal cord paralysis. Although dexmedetomidine (DEX) may be an excellent candidate as a sedative for patients with MSA during spinal anesthesia because of its minimal depression of the respiratory function, no case reports have been published on this topic in the medical literature.

**CASE REPORT**

A 67-year-old woman was scheduled to undergo bipolar hip arthroplasty for left femoral neck fracture. She had been suffered from gait disorder, dysarthria, incomplete voiding, and constipation for three years. On admission, she showed mild orthostatic hypotension: her blood pressure (BP) decreased from 188/79 to 152/93 mmHg without any change in the heart rate (HR) on standing up from a supine position. Her symptoms, cerebellar dysfunction on neurological examination, and atrophy of the brain stem and cerebellum on magnetic resonance imaging (MRI) were all consistent with the diagnosis of MSA. She was so anxious about surgery that she requested sedation during the operation. As we considered that general anesthesia might result in postoperative respiratory complications such as vocal cord paralysis and sleep apnea syndrome (SAS), we planned spinal anesthesia with sedation using DEX. In the operating theater, her BP, HR, and percutaneous arterial oxygen saturation ($\text{SpO}_2$) were 171/78 mmHg, 68 bpm, and 98%, respectively. Spinal anesthesia was performed with 11 mg of 0.5% hyperbaric bupivacaine, and anesthesia was achieved to T10, being sufficient to start the operation. Five minutes after the infusion
of DEX at a dose of 0.33 µg/kg/hr (loading dose), her Ramsay sedation score (RSS) had increased to 4, and the dose was subsequently decreased to 0.69 µg/kg/hr (maintenance dose). Because her BP and HR decreased from 172/78 to 110/57 mmHg and from 68 to 52 bpm, respectively, after the start of DEX infusion, the maintenance dose was titrated to 0.4 µg/kg/hr. The dose was further titrated to 0.28 µg/kg/hr thirty minutes after that, as her BP was 93/51 mmHg. Her hemodynamic state remained stable throughout the operation, without the necessity of any cardiovascular drugs.

She showed no signs of airway obstruction and her SpO$_2$ was maintained above 96% on room air during sedation. The operative time was 72 min. Five minutes after stopping DEX infusion when the operation finished, her RSS recovered to 2. She slept well, remembered nothing about the operation, and was satisfied with the procedure. She was discharged from the hospital 28 days after the surgery without any complications, and her MSA-associated symptoms remained unchanged.

DISCUSSION

This is the first published case report describing the safe and effective use of DEX for sedation of a patient with MSA during spinal anesthesia. MSA is a progressive neurodegenerative disorder characterized by extra-pyramidal symptoms, cerebellar ataxia, and autonomic dysfunction. MSA patients sometimes have respiratory complications such as vocal cord paralysis and SAS. In those with autonomic failure, their ventilatory responses to both hypoxia and hypercapnia have been reported to be markedly impaired, and they are also highly sensitive to tranquilizers and opioids. Those findings led us to select spinal rather than general anesthesia. Our patient wanted to be sedated during the operation since she was very anxious about being alert in the operating theater. Among the sedatives available, DEX has some notable advantages, which are markedly different from those of GABAergic sedatives such as propofol and benzodiazepines. An electroencephalogram (EEG) taken during DEX-induced sedation resembles that of normal sleep. As a result, the muscle tone including that of respiratory muscles is well-preserved, and so its administration does not cause respiratory depression. The present patient showed no signs of airway obstruction and her SpO$_2$ was maintained above 96% in room air during sedation.

Autonomic dysfunction, one of the major symptoms of MSA, may lead to orthostatic hypotension due to a degenerated nucleus of the sympathetic system and/or unpredictable responses to vasopressors due to denervation hypersensitivity of the sympathetic system. A case report demonstrated that patients with an already degenerated sympathetic system were unlikely to become more hypotensive on performing sympathetic blockade with spinal/epidural anesthesia. We selected spinal anesthesia with hyperbaric bupivacaine to easily control the level of anesthesia to as low as possible to perform the operation without pain.

DEX itself may cause cardiovascular problems because it can induce unpredictable changes such as hypertension, hypotension, and bradycardia, especially on high-dose infusion. Although DEX infusion for a patient with MSA might be challenging, its safe use was reported in an infant with familial dysautonomia. In our case, reduction of the DEX loading dose and titration of the maintenance dose were sufficient to maintain a stable BP and HR without requiring any cardiovascular drugs.

As a result, DEX infusion for sedation during spinal anesthesia may be a safe and effective option for patients with MSA. The accumulation of case reports is required to more definitively investigate the potential impact of α2-adrenoceptor agonists on patients with autonomic dysfunction.

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YO, LN — reviewed the manuscript
TO — supervised the anesthetic management and reviewed the manuscript
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REFERENCES