

Epidural anesthetic management using ropivacaine in a parturient with multi-minicore disease and susceptibility to malignant hyperthermia

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To the editor: A 32-year-old woman was admitted to our hospital for delivery of her second child, at 37 weeks' gestation. Details of the delivery of her first child were reported by Osada et al. [1]. In brief, the woman had been a floppy infant at birth, and hypotonus had been recognized when she was an infant.

At 24 years of age, during her first pregnancy, she had been referred to a neurologist because neuromuscular examinations showed marked weakness and atrophy of muscle groups in the trunk and proximal extremities. Her muscles had revealed susceptibility to fulminant malignant hyperthermia (MH), and multi-minicore disease (MmD); her mother had had similar findings. Her laboratory evaluation had been normal and creatinine kinase activity was not elevated. Her first pregnancy had resulted in a normal delivery, with epidural anesthesia using bupivacaine [1].

At the present admission, she was scheduled for an induced vaginal delivery at 37 weeks, with epidural anesthesia using ropivacaine. An epidural catheter was inserted at the L3/4 interspace on the day before the day of delivery. A test dose of 0.2% ropivacaine was titrated and a total dose of 4 ml was given. During this time, we monitored her heart rate (HR), blood pressure (BP), oxygen saturation ($S_{p_{O_2}}$), and body temperature, but no abnormal signs were seen. The next day, labor was induced using oxytocin, and the epidural anesthesia provided good pain relief, with 0.2% ropivacaine (10–16 mg·h⁻¹) and fentanyl (0.01–0.016 mg·h⁻¹) through the epidural catheter. During the labor, BP, HR, $S_{p_{O_2}}$, and body temperature were strictly monitored every 10 min. After 12 h of labor, a 2940-g female infant was born, with Apgar scores of 8 and 9 at 1 and 5 min, respectively. The total dose of ropivacaine was 184 mg and that of fentanyl was 1.8 mg. She and her newborn left the hospital uneventfully 4 days later.

MmD is a type of congenital myopathy. Inheritance can be autosomal dominant, and the clinical course can be mild and nonprogressive, with generalized weakness and hypotonia beginning in infancy, and delayed motor development. The

relationship between MmD and ryanodine receptor or MH is unclear [2].

To reduce the possibility of an MH episode in this patient, we planned epidural anesthesia because it can reduce the stress of labor. It is known that ropivacaine has advantages over bupivacaine in terms of cardiovascular and central nervous system toxicity.

A multicenter, randomized, controlled trial showed that, in labor, there was no difference in obstetric or neonatal outcomes—but there was a significant reduction in motor block—with the use of ropivacaine compared with bupivacaine [3]. So, we used ropivacaine on this occasion, thinking that it was much the better anesthetic for this patient with muscle weakness.

The use of ropivacaine in patients with MH has not been discussed extensively. Maemura [4] reported that ropivacaine accelerated the Ca-induced Ca release (CICR) rate at serum concentrations of 10 mM; however, a clinical dose with a serum concentration of 3 mM is not high enough to accelerate the CICR rate. Maemura's report suggested that ropivacaine could be used safely in patients susceptible to MH, and we informed the patient of this finding.

Ropivacaine could be an acceptable anesthetic for patients with MH, and parturients with MH can be delivered safely with this epidural anesthesia to reduce the stress of labor and to reduce motor block.

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