CASE REPORT

Prolonged sensory impairment in the perineal region after painless delivery through lumbar epidural anesthesia

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Key words
cauda equina syndrome, epidural lumbar anesthesia, neurotoxicity, painless delivery, ropivacaine.

Abstract
In the painless delivery, the neuronal injury caused by regional anesthesia sometimes causes diagnostic difficulty because of the common findings with lumbosacral injury due to vaginal delivery. We herein report a 36-year-old woman presenting with long-standing cauda equina syndrome due to neurotoxicity of regional anesthetic. Neurological examination revealed the almost symmetrical, heterogeneous sensory deficit mainly in the perineal region, hypoactive quadriceps femoris reflex, decreased anal sphincter reflex, and dermographia. The correct diagnosis is important to support the quality of life of patients.

Introduction
Painless delivery is becoming popular worldwide. It provides relief from unbearable labor pain during delivery. Neurological complications after obstetric regional anesthesia are rare, but can occur.1 Perioperative nerve injuries manifesting as cauda equina syndrome (CES) are caused by the epidural anesthesia.2 However, the correct diagnosis is difficult because nerve injuries due to local anesthesia share many neurological symptoms in common with injuries mechanically caused by vaginal delivery itself, which in fact account for the majority of postpartum neuropathies. We herein report a patient with sensory impairment in and around the perineal area caused by the regional anesthetic applied for painless delivery.

Case report
A 36-year-old woman presented with decreased sensation in the perineal region after the first obstetric delivery under lumbar epidural anesthesia. The delivery was uneventful with head presentation. In total, 250 mg of 0.1% ropivacaine and 540 mg of fentanyl were continuously injected through a cannula during the labor and delivery that took 19 h. The patient felt no shooting pain or unusual sensory symptoms during the anesthetic procedure. After the end of delivery, she noticed decreased sensation in the perineal region, and it has been unchanged for a year. Sexual feeling was also lost. She had no problem in urination or defecation.

Neurological examination revealed no muscle weakness in the lower limbs. Pain sensation was moderately and tactile sensation was mildly decreased symmetrically in the perineal region, inguinal regions, buttocks, and lateral and posterior aspects of the thighs. The peri-anal region was spared. The boundary between the hypoesthetic region and the normal region was unclear (Fig. 1). The quadriceps femoris reflexes were weak bilaterally, and only occasionally elicited by Jendrassik maneuver. Ankle jerks were preserved bilaterally. The anal sphincter reflex was weak and slowly reactive. Dermographia was elicited by scratching on the anterior to lateral aspects of the thighs, but there was no abnormality in the pilomotor reflex, moisture, and surface temperature of the skin. Magnetic resonance imaging of the lumbar spine showed no abnormality.

Discussion
The subjective sensory symptoms of the patient were mainly localized to the perineal region, but the sensory deficit was more widely and heterogeneously distributed.

When examining a post-traumatic patient presenting solely with sensory symptoms, possible involvement of a psychogenic factor must be considered. In the present case, however, a hypoactive quadriceps femoris reflex, decreased anal sphincter reflex, and dermographia suggested the presence of an organic lesion involving the cauda equina.

Common neurological complications of vaginal delivery are transection, traction, compression, or vascular injuries of...
the intrapelvic nerves such as the lumbosacral nerves, lateral cutaneous nerves, femoral nerves, and obturator nerves, or compression of the common peroneal nerves at the head of the fibula. Neurological symptoms of lumbosacral nerve injuries in the pelvic cavity overlap considerably with those of CES due to spinal or epidural anesthesia, making the differentiation difficult. In the present case, however, the relatively widespread and symmetric sensory involvement and obscure boundary between the affected and intact dermatomes suggested chemical damage due to the epidural anesthetic rather than the mechanical damage such as traumatic injury by the needle or compression in the pelvic cavity. Dermographia was the only suggestion of automatic involvement. Basically, the pilomotor reflex and the moisture and surface temperature of the skin are innervated by the sympathetic nerve. In contrast, dermographia is usually observed in the allergic disorders such as urticaria, where the vasomotor component of the parasympathetic nervous system is supposed to play a main role. Therefore, the sole emergence of dermographia may not be inconsistent with other findings in the present case, although the underlying mechanism as to how dermographia emerged remains unsolved.

Administration of epidural anesthesia carries a 0.23 (0.01–1.21)/10 000 risk of developing CES. Chemical nerve injury is caused by the toxicity of the applied agents. Reported cases of neurotoxic CES have been mainly caused by lidocaine, bupivacaine, or tetracaine. Neurotoxic injuries caused by ropivacaine, which was used in the present case, have also been reported, but they are relatively rare. In vitro models have shown ropivacaine neurotoxicity in a dose-dependent manner. Ropivacaine is known to block the motor fibers less than the sensory fibers compared to bupivacaine at the same concentration. This might explain the sensory-dominant symptoms in the present case. In terms of the patient-related factors, preexisting systemic diseases such as diabetes mellitus and vascular risk factors are known to increase the risk of peripheral nerve injury.

In the present case, whether the anesthetic insult occurred in the epidural space or intrathecal space remains unclear. Difficulty in the sexual life might have decreased the overall quality of life. The patient is now under periodic follow-up with counseling.

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