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Anesthetic management of a patient suspected of having Lambert-Eaton syndrome due to an unexpected prolongation of vecuronium

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Introduction

Lambert-Eaton syndrome is characterized by a disorder of neurotransmission which is associated with lung cancer or autoimmune diseases [1]. Patients with this syndrome are hypersensitive to nondepolarizing neuromuscular blocking agents during general anesthesia [2]. In the present report, we describe a patient without clinical manifestations of Lambert-Eaton syndrome, who showed unexpected hypersensitivity to vecuronium and was diagnosed as having this syndrome postoperatively.

Case report

A 66-year-old man, weighing 46 kg and 157 cm in height, was admitted to our hospital because of deterioration of cerebellar ataxia which had occurred 3 years earlier. During the 3 years, he became unable to walk straight or eat without assistance. Except for his cerebellar ataxia, there were no neurological symptoms such as muscle weakness or sensory loss. Hematological studies and arterial blood gas analysis were within the normal range. He had no apparent renal or liver dysfunction. Respiratory function was normal: % vital capacity (%VC) was 111% and $FEV_{1.0}$ was 78%. An electromyogram (EMG) was not done preoperatively. He was diagnosed as having late cerebellar cortical atrophy due to progressive cerebellar ataxia and marked cerebellar atrophy on brain computed tomography (CT). On a chest roentgenogram, a coin lesion was unexpectedly found in the right upper lung, for which a transbronchial lung biopsy was performed but no malignant cells were found. An open lung biopsy was scheduled under general anesthesia.

After induction of anesthesia with 250 mg of thiopental and 8 mg of vecuronium intravenously, his trachea was intubated. Anesthesia was maintained with nitrous oxide (60%) and isoflurane (1.0%–1.5%) in oxygen. No neuromuscular blocking agent was administered after the induction, and the patient was hemodynamically stable during the 3-h operation. Antibiotics were not administered pre- and intraoperatively. After the discontinuation of the anesthetics and ventilation with 100% oxygen for over 30 min, his spontaneous respiration did not resume. Although 2 mg of neostigmine was injected, 4 h after the administration of vecuronium, there was no response to twitch or tetanus stimulation by a nerve stimulator. The body temperature of this patient was 36.5°C and the serum concentrations of Ca^{2+} and Mg^{2+} were within the normal range. After another 3 h of mechanical ventilation, a train of four ratio reversed to 100% and his spontaneous ventilation became sufficient. We tested the function of the neuromuscular junctions by an electromyograph (MS-6, Medelec, London, UK). The EMG showed a "waxing phenomenon", in which a 13.5% decrease in amplitude by 32 times of 5 Hz stimulation and a 70% increase by 32 times of 50 Hz stimulation were shown. Antibody against acetylcholine receptor was undetectable in the serum. He was diagnosed as having Lambert-Eaton syndrome. Histologically, he was found to have an oatcell carcinoma of the lung.

Discussion

The pathophysiology of Lambert-Eaton syndrome was elucidated by Lambert et al. as well as by several other investigators [3–6]. Characteristic findings include an

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abnormal low amplitude of the muscle action potential evoked by a single nerve stimulus in a resting muscle and a progressive increase during high-frequency stimulation (>10 Hz) or immediately after a brief maximal contraction of the muscle ("waxing phenomenon") [4]. This abnormality is caused by a decrease in the amount of acetylcholine released from motor nerve terminals by each nerve impulse [7]. Recent reports have shown that acetylcholine release is decreased due to the interference of presynaptic voltage-dependent Ca^{2+} channels by serum IgG of the patient [7–12]. Clinical features [5] include proximal weakness, especially in the lower limbs, with diminished tendon reflexes and posttetanic potentiation by EMG [6]. There are some patients with this syndrome who complain of autonomic features such as dry mouth, impaired lacrimation, and impaired sweating. This may imply a selective cholinergic dysautonomia. The patients with Lambert-Eaton syndrome are hypersensitive to nondepolarizing neuromuscular blocking agents, and the reversal of the effect of these agents by neostigmine is not effective [13–15]. There has been only one reported patient with this syndrome who was given anesthesia who was also free of clinical features [16].

Because the only clinical symptom of this patient was cerebellar ataxia, preoperative diagnosis of Lambert-Eaton syndrome was not made in this patient. The effect of vecuronium was unexpectedly prolonged during the anesthesia. Several causes, such as hypothermia, effects of antibiotics, serum electrolyte abnormalities, and renal or liver dysfunction have been reported to be responsible for the prolongation of the effect of nondepolarizing neuromuscular blocking agents [17]. All of the above factors were ruled out in this case. Myasthenia gravis was unlikely because of the absence of acetylcholine receptor antibody in serum. Therefore, we suspected a disorder of the neuromuscular transmission and made a diagnosis of Lambert-Eaton syndrome based on the EMG findings.

Hypersensitivity to nondepolarizing neuromuscular blocking agents has been reported in patients with the Lambert-Eaton syndrome. Stephen et al. reported hypersensitivity to curare on EMG in a patient without clinical features of Eaton-Lambert syndrome [16]. Although the present patient had no clinical features of Lambert-Eaton syndrome, we experienced the prolongation of the effect of vecuronium. Preoperative EMG might have been a good tool to predict hypersensitivity to nondepolarizing neuromuscular blocking agents.

In conclusion, we reported a general anesthesia for a patient having Lambert-Eaton syndrome without neurological symptoms. Anesthesiologists need to be aware that a prolongation of nondepolarizing neuromuscular blockade may occur during anesthesia for lung cancer.

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