BRADYCARDIA, HYPOTENSION AND BRONCHOSPASM FOLLOWING REMIFENTANIL-PROPOFOL IN A MYATHENIC PATIENT TREATED BY PYRIDOSTIGMINE
- A Case Report -

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Myasthenia Gravis is an autoimmune neuromuscular disorder of skeletal muscles due to the presence of acetylcholine receptor antibodies. Myasthenic patients are resistant to depolarizing relaxants such as succinylcholine but are very sensitive to non-depolarizing muscle relaxants\(^1\). That’s why, narcotic-based technique for induction and maintenance of anesthesia without muscle relaxants has been recommended in myasthenic patients undergoing thymectomy\(^2\). The present report shows that total intravenous anesthesia using a combination of remifentanil-propofol can precipitate severe bradycardia, hypotension and bronchospasm in a myathenic patient receiving pre-operative pyridostigmine therapy up to the morning of surgery.

Case Report

Forty three years old man, 100 kg body weight, having myasthenia gravis was scheduled for trans-sternal thymectomy. The patient was classified as Osserman class I on the basis of having diplopia only without any other muscular weakness. A spiral computed tomography showed an
increased density of adipose tissue localized in the anterior mediastinum. The patient was treated with pyridostigmine 180 mg daily up to the morning of surgery. Preoperatively the HR was 70 bpm and the BP 120/70 mmHg.

In the operating room the patient received glycopyrrolate 0.2 mg IV. Total intravenous technique of anesthesia was then induced using lidocaine 100 mg IV with propofol 2.5 mg/kg followed by remifentanil 2 µg/kg over 60 seconds. Orotracheal intubation was performed without difficulty. Following induction of anesthesia and tracheal intubation, the patient developed severe bradycardia, down to 40 bpm, and hypotension, down to 70 mmHg, associated with bronchospasm with an increase in airway pressure up to 50 cm H₂O. IV injection of glycopyrrolate 0.2 mg and ephedrine 20 mg succeeded to increase the HR up to 80 bpm and BP up to 140/80 mmHg. The bronchospasm was relieved by two intravenous doses of nitroglycerine 100 µg as evidenced by decrease of the airway pressure. Anesthesia was maintained with remifentanil 0.2 µg/kg/min and propofol 10 mg/kg/min supplemented with sevoflurane 2% in O₂/air mixture. Intermittent positive pressure ventilation was adjusted to maintain normocapnia as monitored by end-tidal capnography. Neuromuscular transmission was monitored by a train-of-four nerve stimulator (Innervator 252 FISHER & PAYKEL, Healthcare) which revealed the presence of the four twitches throughout the surgery. The intraoperative period was uneventful. The surgeon removed the hyperplastic thymus completely (thymic excentration).

At chest closure, the propofol infusion was stopped, remifentanil rate was decreased to 0.1 µg/kg/min and morphine 0.1 mg/kg IV was administered. The patient was awake and extubated after adequate tidal volume.

Discussion

It is controversial whether anticholinesterase therapy should be maintained or discontinued in myasthenic patients scheduled for surgery. Patients who have mild symptoms such as localized ocular myasthenia
(Osserman I) or mild generalized myasthenic without bulbar symptoms (Osserman IIa) can interrupt their regimen. In contrast, patients who are dependent on anticholinesterase therapy for their well-being (Osserman IIb, III & IV) should maintain their anticholinesterase therapy till the morning of surgery.

Our report shows that myasthenic patients pretreated with the anticholinesterase pyridostigmine which has a muscarinic effect can develop severe bradycardia, hypotension and bronchospasm whenever anesthesia is induced by large doses of remifentanil-propofol. However, the degree of hypotension, bradycardia and bronchospasm may be accentuated by the preoperative preparation of our myasthenic patients by the large dose of the anticholinesterase pyridostigmine which was maintained up to the morning of surgery. This can be attributed to a summation of the vagotonic effects which are precipitated by the combination of the muscarinic effect of pyridostigmine and the central vagotonic and/or sympatholytic effect of remifentanil and propofol.

Severe bradycardia has been previously reported following propofol with fentanyl, succinylcholine. Because of the possible potentiation of the central vagotonic effect of propofol and remifentanil by the peripheral muscarinic effect of pyridostigmine, it may be advisable to withhold pyridostigmine the morning of surgery in myasthenic patients classified as Osserman I or IIa. However, pyridostigmine therapy must be continued till the morning of surgery in myasthenic patients classified as Osserman IIb, III, or IV. In this situation, anticholinergic medications such as glycopyrrolate must be administered prior to induction of anesthesia by remifentanil-propofol combination. Also, further doses of anticholinergics or sympathomimetic drugs such as ephedrine should be administered intravenously whenever bradycardia and/or hypotension follows induction of anesthesia.
References