



Research Paper

Anesthetic Management Of A Patient With Myasthenia Gravis For Thymectomy - Case Report

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I. Introduction:-

Myasthenia gravis (MG) is either an autoimmune or congenital neuromuscular disease that leads to fluctuating muscle weakness and fatigue. In the most common cases, muscle weakness is caused by circulating antibodies that block acetylcholine receptors at the postsynaptic neuromuscular junction [1], inhibiting the excitatory effects of the neurotransmitter acetylcholine on nicotinic receptors at neuromuscular junctions. Alternatively, in a much rarer form, muscle weakness is caused by a genetic defect in some portion of the neuromuscular junction [2]. The disease incidence is 3–30 cases per million per year and rising as a result of increased awareness. Medical treatment of MG includes improving neuromuscular transmission by anticholinesterases, suppressing the immune system with corticosteroids and immunosuppressants, and circulation of antibodies with plasmapheresis and, in selected cases, thymectomy [3]. Patients with generalized MG and patients with ocular symptoms poorly controlled by anticholinesterases often benefit from thymectomy [4].

The myasthenic patient can be a challenge to anesthesiologists, and the post-surgical risk of respiratory failure has always been a matter of concern. During postoperative period pain, analgesics and residual effects of anaesthetics can adversely affect pulmonary function which is already limited by the MG itself. This makes it important for the anesthesiologist to be aware of possible signs of disease and to be properly updated on the optimal perioperative anesthesiological management of the myasthenic patient [5]. This report describes the management of anesthesia in a patient with MG undergoing thymectomy.

II. Case Report:

The patient was a 51 yr-old male, presented with complaints of diplopia and ptosis, 3 years back and was diagnosed with oculobulbar myasthenia gravis. On evaluation, a thymic mass was detected and thymectomy was planned. Patient was taking Tab. Pyridostigmine 50 mg twice daily and Tab. Deflazacort 12 mg once daily.

Preoperative blood investigations, respiratory, cardiac and thyroid functions were normal. Written informed consent was obtained from patient. ECG, invasive arterial pressure, pulse oximetry, capnography and central venous pressure were continuously monitored. With patient in right lateral, epidural puncture was performed from a median approach with a 18 G Tuohy's needle between T6 -T7 vertebrae. The epidural space was identified by loss of resistance to air, and catheter was threaded into the epidural space. Epidural test dose was given with 3 ml Inj. Lignocaine 1.5% with Adrenaline 1 in 2,00,000. The sensory level was obtained between T2-T6.

Anaesthesia was induced after preoxygenation with 2 mcg/kg bolus Fentanyl, 1.5 mg/kg lignocaine and 2 mg/kg bolus propofol and tracheal intubation was done with left sided double lumen endotracheal tube for one lung ventilation. After the tracheal intubation, fentanyl was infused 2 mcg/kg/hr dosage. The patient was ventilated with a 50% mixture of oxygen and air to maintain end tidal carbon dioxide between 30 and 35 mm Hg. Inj. Propofol injection at 100 mcg / kg/min was started. Left lung ventilation was applied during surgery via left side double lumen endotracheal tube. The patient was hemodynamically stable during intubation and the surgical procedure. In the first hour and second hour of the surgical procedure, the patient received Inj. Bupivacaine 0.25% 12 ml and 8 ml via the epidural catheter.

A continuous epidural infusion of Bupivacaine 0.125% was administered through the epidural catheter for postoperative pain management. At the end of surgery, the patient was easily extubated and was transferred to the

intensive care unit. In the ICU, the patient was hemodynamically stable without any subjective or objective impairment of respiratory function.

III. Discussion

The resultant respiratory and cardiovascular implications are a primary cause of mortality; therefore, a complete and comprehensive understanding of this disorder is vital for the anesthesia provider [6].

The use of muscle relaxants in patients with MG has been a controversial topic. Therefore, the anesthesia of patients with MG requires special attention, particularly in respect of the use of muscle relaxants. Current general anesthesia requires volatile anesthetic agents and sometimes muscle relaxants for tracheal intubation and anesthetic maintenance. Patients with MG are usually sensitive to the effects of non-depolarizing muscle relaxants, and volatile anesthetic agents accelerate their effect. Furthermore, the potential interaction of anticholinesterases (administered as therapeutic agents for MG) with both the depolarizing and non-depolarizing muscle relaxants is also a problem. The respiratory insufficiency introduced by such agents and the complications due to endotracheal tube placement can continue postoperatively and may require reintubation or prolonged intubation [7-9].

It is difficult to determine the optimal amount of muscle relaxants required in a patient with MG. Hence, there is an increasing trend of using non-muscle relaxant techniques in such patients who undergo surgery [7-9].

Various anesthesia procedures have been reported for patients with MG, such as the use of combination propofol and fentanyl, sevoflurane and fentanyl, propofol and sufentanil or propofol and remifentanyl without muscle relaxants in the literature. Some studies have also reported the use of TEA in combination with techniques of general anaesthesia [9-13].

TEA suppresses hormonal and metabolic stress response to pain, allowing stable hemodynamics during surgery and good postoperative analgesia without compromising pulmonary function [13].

Responses to endotracheal intubation arise essentially due to sympathetic stimulation causing increases in blood pressure, increases in heart rate and tachyarrhythmia [14]. Opioid narcotics were shown to provide hemodynamic stability and suppress most reactions to surgical trauma. Remifentanyl has an extremely short context-sensitive half time which allows it to be administered in very high doses during particularly stressful intraoperative phases without prolonging recovery time.

In this case, a combination was used of low MAC sevoflurane anesthesia and an infusion of remifentanyl with thoracic epidural anesthesia. Muscle relaxants were not used and hemodynamic responses to intubation were controlled effectively. The elimination of remifentanyl is fast so that is possible to rapidly reduce its concentration in the plasma by changing the speed of infusion. The infusion of remifentanyl and TEA allowed for the adjustment of the depth of analgesia during the operation. Effective analgesia for surgery was achieved in this patient with the use of TEA, but the low MAC sevoflurane anesthesia and infusion of remifentanyl were needed for the patient to be able to tolerate the tracheal tube allowing adequate one lung mechanical ventilation without the use of muscle relaxants.

Clinicians are well aware of the risk of postoperative respiratory failure that may result from stress-induced exacerbation of MG (myasthenic crisis), an overdose of anticholinesterases (cholinergic crisis), the residual effects of myorelaxants or other adverse drug interactions (with antibiotics or antiarrhythmics). Therefore, routine postoperative ventilatory support and planned extubation in the ICU have been recommended for high-risk patients [15].

In conclusion, this combination avoided the use of muscle relaxants or high MAC volatile agents was well tolerated for tracheal intubation and allowed a quick transition to spontaneous breathing and a rapid recovery from anesthesia, good postoperative analgesia and an uneventful recovery.

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