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TREATMENT OF RESPIRATORY INSUFFICIENCY AFTER OPEN HEART SURGERY IN A CASE OF MYASTHENIA GRAVIS

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In myasthenia gravis patients, need for mechanical ventilator support is prolonged especially after open heart surgery because of weakness in respiratory muscles. In such cases, mortality and morbidity is decreased by effective treatment of infections, using corticosteroids and anticholinesterases.

Key words: Myasthenia gravis, open heart surgery, respiratory insufficiency

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There are many clinical situations where mechanical ventilator therapy is needed after open heart surgery. These may be pulmonary or extrapulmonary in origin. In the extrapulmonary group, myasthenia gravis (MG) and myasthenic syndrome cases may cause difficulties in weaning in the postoperative period.

CASE REPORT

A seventy-year-old male patient was admitted to our department for CABG operation. He reported atypical chest pain and difficulty in breathing that has occurred twice for short periods in the history. The preoperative laboratory results were not specific except for negative T waves in ECG (V 1-4) and significant aortic contours in chest x-ray. Operation was started in elective conditions after premedication with midazolam 0.1 mg/kg IM. Etomidate (0.3 mg/kg), fentanyl (5 mcg/kg) and vecuronium bromide (0.1 mg/kg) were used for anesthesia

induction. After intubation, arterial (radial artery) and central venous (internal jugular vein) cannulations were done for invasive blood pressure and central venous pressure monitoring. Fentanyl, midazolam and vecuronium bromide were used for maintenance of anesthesia.

In CABG operation, LIMA-LAD and Ao-D₁-D₂ anastomoses were done. After the operation, the patient was transferred to the intensive care unit. Hemodynamic variables were stable and the patient was extubated 10 hours after the operation with the recovery of his motor functions.

Fifteen minutes after the extubation, the patient began to sweat and had dyspnea. Intubation was necessary because of decreasing SaO₂ values. Extubation was performed again 7 hours later, upon stabilization of the hemodynamic and pulmonary functions. Four hours after the extubation, he developed the same symptoms and was intubated once again. During the following 24 hours, he was time to time connected to the T-system for spontaneous breathing but in every attempt he was tired and connected to the ventilator again. Scopy was performed on the 2nd postoperative day and bilateral diaphragm elevation was observed. Neurology consultation was carried out upon consideration of diaphragm paralysis. When the details of the patient's history were examined, he was found to have an episode of ptosis that has lasted for 3 days 5 years ago. He was suspected to have a neuromuscular disorder and a treatment regimen with mestinon (3x60 mg) + corticosteroids was ordered. Tracheostomy was performed and oral feeding was started. In the following 9 days, spontaneous breathing intervals increased in time. He was disconnected from the ventilator on the postoperative 12th day and taken out from the intensive care unit to the ward with tracheostomy cannula left in place.

For the diagnosis of MG, anticholinesterase antibody level was studied and found to be 7.6 nmol/L (values below 0.2 nmol/L were accepted to be in the normal range). The patient was discharged on the postoperative 18th day.

Myasthenia gravis is not a rare disorder. It has a prevalence of 1:20.000 in the western countries which shows a peak in the 3rd decade in women and 5th decade in men. The M/F ratio is 3/2 (1).

Myasthenia gravis is an autoimmune disease of the neuromuscular junction. The present antibodies react with and block the acetylcholine receptors in the postsynaptic membrane. This blockage results in a decrease of activated receptors in the junction which eventually leads to a weak contraction. Electrophysiological studies showed that in myasthenia gravis patients, the end-plate potentials in the neuromuscular junctions are normal in quantity but have decreased amplitudes (2).

In recent years, the studies showed that thymic lymphocytes change in number and response to mitogens with increasing age and this has a role in the pathogenesis of some autoimmune diseases (3).

The clinical spectrum may vary according to the characteristics of the patient. Most common findings are weakness and getting tired easily. The ocular muscles are usually involved. In 15-20% of myasthenia patients only the ocular and eye-lid muscles may be affected. Variations of diplopia and ophthalmoparesis may be seen. Even though ptosis is common, pupils are never involved (2). In our patient's history, an episode of ptosis was present.

The respiratory muscles are frequently affected. The most common reasons of hospitalization in intensive care units are dyspnea and the problem of oral and respiratory secretions.

As soon as MG is suspected in a patient, some diagnostic tests must be carried out:

1. Tensilon test: edrophonium hydrochloride (tensilon) is a fast, short acting parenteral cholinesterase inhibitor. Weakness in myasthenia recovers transiently after injection of 4-10 mg of tensilon.

2. Anti-acetylcholine receptor antibodies: Except for ocular myasthenia gravis where there are no antibodies, antibodies against acetylcholine receptors are found in approximately 85% of the cases (4). Although the existence of antibodies is a strong evidence

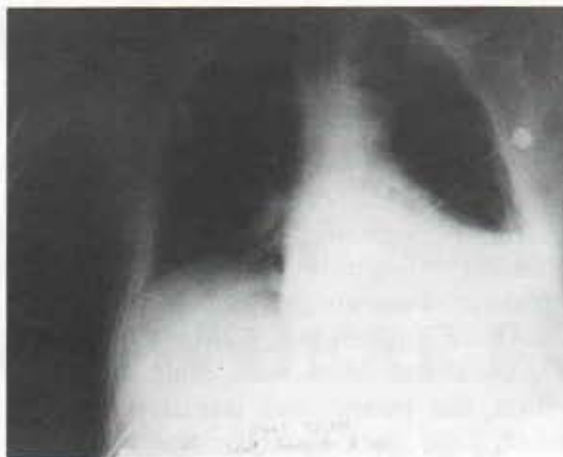
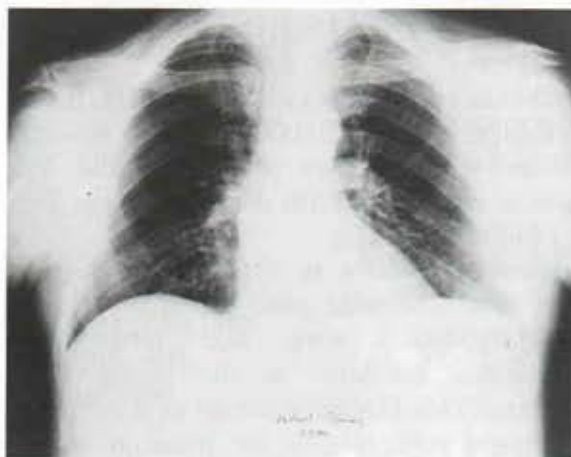


Figure 1. Preoperative (A) and postoperative 3rd day (B) chest x-rays.

for diagnosis, their quantity is not correlated with the severity of the disease. Especially in the patients with ocular symptoms, normal test results do not rule out the diagnosis. In our case, the quantity of acetylcholine receptor antibodies was found to be 7.60 nmol/L (Reference value < 0.2 nmol/L).

3. EMG: EMG measures muscle potential in myasthenia gravis after exercise or slow repetitive nerve stimulation. The decrease should be at least 10%. However required value is 15% or higher. Motor and sensory conductance is normal.

Myasthenia gravis can be concomitant with malignant thymoma or thymic hyperplasia. When diagnosis is made, a thorax CT must be taken since there may be a concomitant thyroid or other autoimmune disease (2,3). Differential diagnosis of myasthenic syndrome (Eaton-Lambert Syndrome) must be made. The patients with this syndrome, which is usually associated with carcinomas or motor neuropathies, are sensitive to depolarizing and non-depolarizing muscle relaxants (5).

The need for surgery in myasthenic patients was a major risk of mortality and morbidity. Today, this risk has become less with the improvement in intensive care techniques and treatment modalities in MG. If not known beforehand, MG must be suspected when the patient can not be disconnected from the ventilator postoperatively. If the patient is known to have the diagnosis, preoperative pulmonary functions must be examined in details. The preoperative status of the patient gives clue about the post-operative period.

Because of myasthenic weakness, diaphragm and accessory respiratory muscles are less efficient when breathing work is increased after extubation (2). In our patient as well, a respiratory weakness has developed which lasted for increasing time intervals upon extubation trials, thus intubation was necessary. Diaphragm was seen to be elevated in the chest x-ray.

Infectious diseases increase muscle weakness in myasthenic patients. For this reason, infections must be avoided in the postoperative period. We used a 2g/day cistatin regimen in addition to routine surgical prophylaxis.

Electrolytes have an important effect on neurotransmitters. In the postoperative period imbalances of especially potassium, magnesium, calcium and phosphorus must be corrected. In myasthenic patients, the drugs that have very little effect on neuromuscular junction may result in exaggerated responses. Lidocaine, quinidine, procainamide, beta-blockers and calcium channel blockers which are commonly used in cardiovascular intensive care should be avoided (2). Verapamil that blocks voltage-dependant calcium channels especially in cardiac and smooth muscles increases the weakness in myasthenia patients (6). Also the opioid analgesics, anticholinergics and most antibiotics commonly used in intensive care may affect neuromuscular junction. These drugs can make postoperative weaning difficult in mild myasthenia cases.

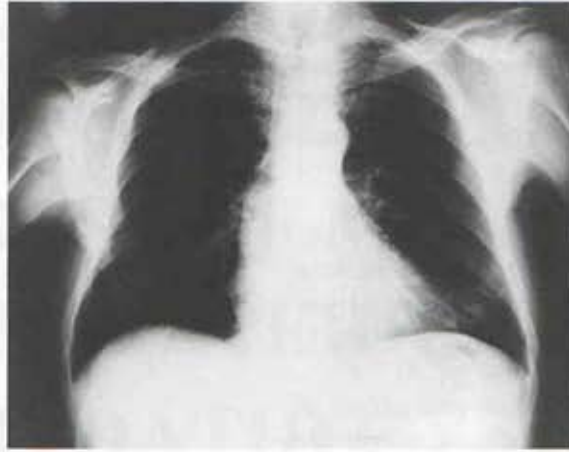


Figure 2. Chest x-ray on the 45th day.

CONCLUSION

Plasmapheresis, corticosteroids and cholinesterase inhibitors are used in the treatment of myasthenia gravis. As response to plasmapheresis begins 48 hours after the procedure, it is not a good choice for postoperative patients. Corticosteroids are used especially in case where low dose cholinesterases are inefficient. They have no common side effects and are useful in more than 80% of the patients. Although a prednisone dose of 25 mg/day is usually effective, it may be increased up to 60 mg/day. Cholinesterase inhibitors are the major treatment modality in intensive care unit. Continuous IV infusion of neostigmine 1 mg is used and after the positive response oral pyridostigmine 60 mg qid should be started. Maintenance dose is 60 mg bid.

After the consideration of myasthenia gravis in our patient, we began the treatment with prednisone 40 mg/day and neostigmine 0.5 mg bid. After tracheostomy, we changed the regimen to oral pyridostigmine 60 mg bid. The patient is still on pyridostigmine treatment 120 mg/day. Control chest x-ray of the patient who was found to have a functional capacity level of 1 (NYHA) is shown in Figure 2.

Respiratory complications are commonly seen in MG patients after surgical treatment. Myasthenia gravis should be investigated in the history of the patients scheduled for a major surgical intervention and suspected in the patients who have problems in weaning postoperatively. In these patients, respiratory insufficiency may become a major factor of mortality and morbidity which is usually minimized with appropriate medical treatment.

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