Case report

An unusual case of multiple failed extubations in a neurosurgical patient

Gana Boban Thadathil*, Jithendra, Mathew George, Jithin K Sree, Binu P J, Jeevan Lal

Email: gana.thadathil@gmail.com

Abstract

A 41 year old woman, ASA PS1, an operated case of Chiari type 1 malformation, syringomyelia and syringobulbia, was posted for right syringopleural shunt. After a routine on-table extubation, immediately postoperatively, she lost consciousness, became progressively hypoxaemic requiring emergent reintubation. Blood gases revealed severe respiratory acidosis. The patient was systematically evaluated for likely causes of respiratory failure considering the sites of surgery and her preoperative surgical condition, *i.e.*, brainstem, chest and larynx. She failed three trials of extubation, at different stages of her evaluation. Suspecting an undiagnosed neurological condition, the neurologist's evaluation discovered a three-month history of easy fatiguability, dysphagia and ptosis. Nerve conduction studies also pointed to the diagnosis of myasthaenia gravis. After initiating steroids and neostigmine, the patient made a steady recovery and was successfully weaned off the ventilator.

Keywords: Myasthaenia gravis, failed extubations

Introduction

Myasthaenia Gravis is an autoimmune disorder characterised by the production of antibodies against one's own nicotinic (postsynaptic nicotinic) acetylcholine receptors. This produces weakness of transmission of impulses across neuromuscular junction (NMJ) and failure to produce an action potential.[1] The number of acetylcholine receptors is also reduced at the NMJ. The prevalence is estimated to be 1:20,000 in the general population.[2] Women are more affected than men.

Gana Boban Thadathil, B.Sc RT
RT Intern

Jithendra, MD
Associate Professor of Anaesthesiology

Mathew George, MD
Associate Professor of Anaesthesiology

Jithin K Sree, B.Sc RT, MSc RT
Chief Respiratory Therapist

Binu P J, B.Sc RT
Respiratory Therapist

Jeevan Lal, B.Sc RT
Respiratory Therapist

Amrita Institute of Medical Sciences, Kochi

The usual clinical presentation is weakness of skeletal muscles, especially with exertion or continued use. With rest, the muscles partially recover unless a myasthaenic crisis is precipitated requiring mechanical ventilation. Skeletal muscles innervated by cranial nerves are especially vulnerable.[3]

Case report

A 41 year old, female patient, diagnosed to have Chiari type 1 malformation with syringomyelia and syringobulbia, with a past history of foramen magnum decompression and syringo-subarachnoid shunt underwent right syringopleural shunt.

The patient was extubated in the operating room soon after surgery after reversing neuromuscular blockade. She was conscious, oriented and haemodynamically stable with normal oxygen saturation on simple face mask 5 L/min of oxygen. Within half an hour after being shifted to ICU, she complained of shortness of breath and was tachypnoeic with oxygen saturation of 90-95%. An arterial blood gas sample (ABG) was taken immediately and the patient was put on noninvasive ventilation (NIV) with an inspiratory airway pressure (IPAP-10 cm H_2O), and expiratory

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positive airway pressure (EPAP-5 cm H_2O), rise time -200 ms). ABG analysis showed mild respiratory acidosis (PCO_2 – 49 mm Hg, PO_2 – 155 mm Hg, pH - 7.24). Patient tolerated NIV for about one hour maintaining normal oxygen saturation. Within the next hour, she became slightly drowsy with altered sensorium and episodes of desaturation (SpO_2 85-92 %). A repeat ABG showed severe respiratory acidosis (pH-7.17, PCO_2-61 mm Hg, PO_2-75.4 mm Hg, HCO_3^- - 22.9 mmol/L).

A portable chest radiograph did not reveal any abnormality. The possibility of opioid-induced respiratory depression was considered but there was no significant improvement in sensorium after naloxone. Residual neuromuscular blockade was also considered for which additional dose of anticholinesterase was given. There was mild improvement in symptoms characterised by improvement in sensorium and respiratory rate. However, within a few minutes, she became drowsy again, was reintubated and ventilated with synchronised intermittent mandatory ventilation (SIMV) with the following settings: tidal volume of 400 ml, respiratory rate 12 breaths/min, positive end-expiratory pressure 5 cm H_2O and 50% oxygen.

An ABG taken after reintubation showed a pH of 7.02, PCO_2 - 98.8 mm Hg, PO_2 -91.4 mm Hg, HCO_3^- 24.4 mmol/L. Further adjustments in ventilator settings were done and subsequently ABG values normalised. The plan was to sedate and ventilate overnight, reassess for weaning and extubate her trachea the next morning.

Sedation was stopped early morning. The patient became conscious, oriented and was obeying verbal commands. She was gradually weaned off from SIMV to pressure support ventilation (PSV). ABG on this setting showed pH - 7.46, PCO_2 - 26.3 mm Hg, PO_2-226 mm Hg, HCO_3^- - 19.2 mmol/L. A spontaneous breathing trial (SBT) with T-piece was given and well tolerated. A decision to extubate and continue with NIV was taken but immediately after extubation, she complained of shortness of breath and with desaturation. Emergency reintubation was done and she was put back on SIMV with adequate sedation. Surgical complication such as inadequate decompression or iatrogenic medullary damage was suspected. A CT and MRI of upper cervical spine was done which revealed good decompression of syrinx. Chest radiograph did not show any evidence of pneumothorax or pleural effusion. A plan to continue ventilation and sedation for another 24 h was made. Blood gas studies revealed normal values.

A repeat SBT trial with PSV and T-piece next morning was well tolerated. She was extubated for the fourth time after confirming normal blood gas values on ABG on T-piece. Postextubation, mild to moderate tachypnea was noted, SpO_2 was 100% on simple face mask with 5L/min of oxygen.

An assessment of upper airways, vocal cords and subglottic region was done with a flexible bronchoscope which showed mild oedema over the supraglottic region, mild inadequacy of left vocal cord movement and vocal cord aperture seemed to be fixed. She was given a trial of NIV for 2-3 hours but developed respiratory distress and became drowsy. An immediate ABG showed respiratory acidosis (pH-7.04, PCO_2 – 126 mm Hg, PO_2 – 105 mm Hg, HCO_3^- - 34.5 mmol/L). She was intubated and planned to continue ventilation for next 48 hours till the cause for respiratory failure could be elucidated.

The differential diagnosis considered at this stage for failure to wean was neuromuscular disorder such as myasthaenia gravis, Guillain Barre syndrome *etc.* A careful history-taking from the relatives revealed that she had symptoms of easy fatiguability, dysphagia, ptosis with diurnal variation since the last 2-3 months. Considering the above symptoms, an evaluation for myasthaenia gravis (MG) was started. Nerve conduction studies showed delayed conduction in all peripheral nerves and right phrenic nerve. Sensory conduction study was within normal limits. Repetitive nerve stimulation (RNS) was done which showed decremental response from ulnar and facial nerves with successive stimulation. Considering the above reports, a CT scan of the chest was taken which revealed minimally enhancing mass lesion in the anterior mediastinum suggestive of a thymic mass. Acetylcholine receptor antibodies and intravenous neostigmine test were positive.

The diagnosis of Myasthaenia gravis was confirmed. She was started on acetylcholinesterase inhibitors (Tab Neostigmine), Tab Azathioprine (immunomodulating drug) and steroids. Mechanical ventilation was continued for two more days after commencing treatment for myaesthenia gravis.

The patient was gradually weaned from SIMV to SBT with PSV and T-piece trial which was well-tolerated. She was then mobilised on and around the bed on T-piece with ET tube *in situ* and supplemental oxygen *(Figure 1)*.

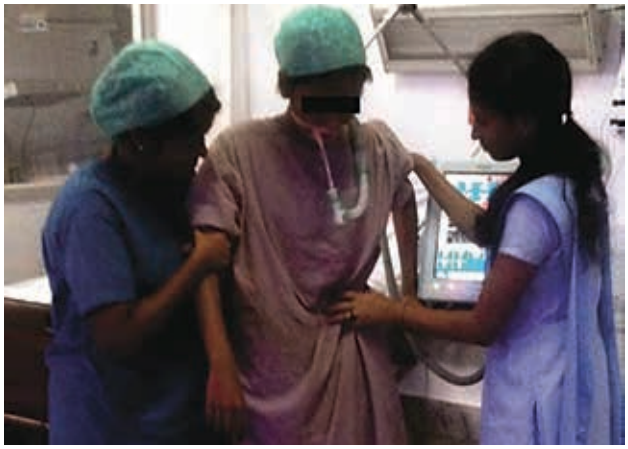

Figure 1: Rehabilitation of the still intubated and mechanically ventilated patient

Multiple SBT trials thereafter never showed any sign of weakness, tachypnoea or desaturation. ABG obtained intermittently during weaning trials were within normal limits. She was extubated and was given intermittent NIV.

NIV was reduced and weaned off gradually with intermittent periods of spontaneous breathing with oxygen through nasal prongs. Mobilisation of the patient while on nasal prongs did not reveal any events of desaturation and tachypnoea. Deep breathing exercises and aerosol therapy were continued. She was gradually weaned off to room air and was shifted from ICU to ward on day 14. In the ward she resumed her normal activities with regular follow up.

Discussion

Undiagnosed myasthaenia gravis in patients undergoing surgery under general anaesthesia can lead to repeated failed extubations and ventilator dependence in the postoperative period. In this patient, the myasthaenia symptoms which were present before the second surgery may have been mistaken to be secondary to the cervical syringomyelia and syringobulbia. A retrospective analysis of preoperative symptoms led to the evaluation and diagnosis of MG in the patient. She did not have the myasthenic symptoms prior to the first surgery and hence went undiagnosed. She underwent thymectomy two months later while on treatment for MG. She had an uneventful postoperative recovery and was extubated within 24 hours after surgery with continuation of neostigmine, steroids and azathioprine in the perioperative period. This case emphasises the need for considering associated neuromuscular disorders that can complicate postoperative recovery of patients with failed extubations and weaning from ventilator.

Myasthaenia gravis is an autoimmune disorder due to development of antibodies directed to nicotinic receptors at the muscle end plate. Reduction in the number of free neuromuscular cholinoceptors and structural damage to the neuromuscular junction lead to weakness and easy fatiguability on repeated activity with recovery after rest. MG can be treated with cholinesterase inhibitors such as neostigmine, pyridostigmine and immunomodulating drugs such as azathioprine. Depending on severity of the condition, NIV or invasive ventilation has a major role. Some of them may require long term ventilatory support and will benefit from appropriate and protocolised use of NIV.[4]

References

1. Baraka A. Anaesthesia and myasthaenia gravis. *Can J Anaesth* 1992; **39**(5): 476-86
2. Drachman D B. Myasthaenia gravis. *N Engl J Med* 1978; **298**:136-42
3. Hughes BW, De Casillas MLM, Kaminski HJ Pathophysiology of myasthaenia gravis. *Semin Neurol* 2004; **24**:21-30
4. Bates DV, Christie RV. Respiratory function in disease – An introduction to the integrated study of the lung. WB Saunders, Philadelphia. 1989. 3rd edition.