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Undiagnosed Myasthenia Gravis Presenting With Decreased Sensorium and Carbon Dioxide Narcosis

Case Report

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Abstract

When a patient comes to the emergency department with decreased sensorium, a thorough examination should be done in order to arrive at a proper diagnosis and treatment. This includes evaluation of cerebral vascular disorders like haemorrhage and infarct; metabolic and electrolyte abnormalities like hypoglycaemia, hyponatremia, hypercalcaemia, uraemic and hepatic encephalopathy; hypoxia; carbon dioxide narcosis; and carbon monoxide poisoning. We recently encountered an elderly male patient who came to the emergency department with unconsciousness and was found to have a raised carbon dioxide level in the blood and, subsequently, a high level of antiacetylcholinereceptor antibodies. The patient was treated in line for myasthenia gravis, following which he made a complete recovery.

Learning Points

- Myasthenia Gravis can present with decreased sensorium as an initial presentation.
- Myasthenia Gravis should be considered in any patient with unexplained difficulty weaning from ventilator support.
- High degree of suspicion and early diagnosis are required for a better outcome. Myasthenia Gravis, decreased sensorium, carbon dioxide narcosis.

Keywords: Myasthenia gravis;Decreased sensorium; Carbon dioxide narcosis

Introduction

Myasthenia gravis is a chronic autoimmune condition affecting the neuromuscular junction. Though the disease is characterised by weakness and fatigability of skeletal, ocular, and bulbar muscles, the disease may also present with carbon dioxide as an initial manifestation. Here, we present the following case:

Report a case

A 74-year-old male patient with a known case of diabetes mellitus, hypertension presented to the emergency department with difficulty in breathing for 3 days, followed by unconsciousness for 1 day. According to the patient's attendant, there is no history of fever, cough, chest pain, or seizure, nor is there a history of alcohol, smoking, or substance abuse.

On arrival in the emergency department, the patient was unconscious with GCS of E1V1M1-3/15, afebrile, pulse-120 beats per minute, B.P.-130/80 mmhg, respiratory rate-20 breaths per minute, and chest-bilaterally vesicular breath sound. CVS-No abnormality detected. Pupil size was 3 mm bilaterally with a sluggish reaction to light. The plantar reflex was bilaterally extensor. SpO2 of 94 percent in room air, RBS-253 mg/dl.

The patient was intubated and connected to a mechanical ventilator because of low GCS (3/15) and then shifted to the ICU.

An MRI of the brain showed age-related cerebral degenerative changes.

Routine investigation of blood revealed TC-11800/cumm. Hemoglobin-13gm/dl, ESR-20mm, Na-143.1mg, K-4.24mg/dl, Ca-8.24mg/dl, Mg-1.95mg/dl, Abg-Ph-7.35, Po2-70mmhg,. Pco2-86mmhg, HCO3-28mmol, TSH-3.22mIu/L,

A chest X-ray revealed no signs of consolidation, infiltrate, or pleural effusion.

The CT thorax revealed mild interstitial edema but no lung consolidation or mass. ECG-sinus tachycardia, Troponin I-negative. Echocardiography findings are of concentrated left ventricular hypertrophy and grade 1 diastolic dysfunction with normal left ventricular systolic function.

From the history, physical examination, blood investigation, and imaging study, the initial impression we got was that Chronic Obstructive Pulmonary Disease (COPD) was presenting as carbon dioxide narcosis and the carbon dioxide level was very high.

The patient was treated with intravenous fluids, broad-spectrum antibiotics, bronchodilators, low tidal volume controlled ventilation (VC)-Tidal volume-360 (60kg), Peep-3 cm water, RR-14, Fio2-40%.

However, despite improvement of sensorium over the next few hours of ventilator support, the patient could not be weaned from the ventilator as he became drowsy with reduced spontaneous respiratory rate and a rise in arterial carbon dioxide level as the ventilator support was reduced.

A neurological condition consultation was taken in order to rule out the neurological condition of hypercapnia. The diagnosis of Guillian-Barre syndrome was not considered because the patient had no history of lower limb weakness and the plantar was bilaterally extensor with the presence of a deep tendon reflex. Next, for the diagnosis of myasthenia gravis, 0.5mg of neostigmine was given IM and an anti acetylcholine receptor antibody was sent. Spontaneous respiratory activity, which was absent initially, was observed at 15 minutes with an increase in respiratory rate from 12 breaths/minute to 24 breaths/minute and a rise in tidal volume from 360 ml to 440 ml. After 45 minutes, a spontaneous respiratory effort stopped and mandatory ventilator breath started. The anti-acetylcholine receptor antibody that was sent came to be highly positive. With the diagnosis of myasthenia gravis confirmed, the patient was treated with a tablet of pyridostigmine (60mg) every four hours,along with other supportive medications. The patient made a dramatic recovery with improvement of sensorium, serial ABG, and increased spontaneous breathing activity and tidal volume and was successfully liberated from the ventilator over the next 48 hrs. Presently, the patient is on regular follow-up on a long-term tablet of oral pyridostigmine and immunosuppressive medication.

Case discussion

Anti-acetylcholine receptor antibodies in the neuromuscular junction cause skeletal muscle weakness and fatigability in myasthenia gravis [1,2]. Myasthenia gravis occurs at all ages, though females are commonly affected in their third decade of life and males in their sixth or seventh decade [3]. The disease is characterized by Ptosis, diplopia, and easy fatigability on repeated use of skeletal muscle [4].

In two-thirds of cases, bulbar and ocular symptoms such as dysphagia, increased oropharyngeal secretions, jaw and tongue weakness, and ptosis mimicking cerebrovascular accident predominate, especially in the elderly group of patients [5]. This, in turn leads to an increased likelihood of micro-aspiration, atelectasis, upper airway resistance, dead space, and the work of breathing, causing ventilation- perfusion mismatch, hypoxaemia, and hypercarbia.

In the following case, a patient presented to us unconscious with no neurological symptoms associated with myasthenia gravis, such as fatigability, ptosis, dysphagia, or increased oropharyngeal secretions. So, initially, conditions like CVA and metabolic and electrolyte abnormalities were ruled out, and arterial blood gas revealed raised carbon dioxide without hypoxaemia. To further explain the cause of the raised carbon dioxide level, we looked for evidence of COPD, pneumonia, pleural effusion, consolidation, pulmonary thromboembolism, or intracardiac shunting, drug intoxication, through appropriate imaging, CT scan thorax, and echocardiography. But it did not reveal any significant abnormality to explain the cause of respiratory failure.

It was only when the patient again required re-intubation and ventilation that the possibility of a neuromuscular disorder was considered, which was confirmed by a positive anti acetylcholine receptor antibody level. Following the administration of anti-acetylcholinesterase medications, the patient made a dramatic recovery with an increase in spontaneous respiratory activity and tidal volume and was successfully weaned from the ventilator and discharged.

Conclusion

Myasthenia Gravis should always be considered in any patient with unexplained altered sensorium, patients who require prolonged ventilator support, and in patients in whom ventilator weaning has failed so that appropriate therapies can be targeted.

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