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# Unusual presentation of acute respiratory failure in myasthenia gravis as organophosphate poisoning in the emergency room

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## ABSTRACT

Myasthenia gravis (MG) is a specific autoimmune disease characterized by weakness and fatigue. It is often complicated by respiratory failure, known as a myasthenic crisis. Most of the patients who develop respiratory symptoms do so during the late course of disease and have other neurological signs and symptoms. However, in some patients respiratory failure is the initial presenting symptom.

We report a case of 18 year old male with no past medical history presented to the emergency room (ER) with signs and symptoms of organophosphate poisoning. Patient subsequently went into acute respiratory failure. He was found to have myasthenia gravis with isolated respiratory failure as his first presenting symptom. As illustrated by this case, it is important to consider neuromuscular disorders in cases of unexplained respiratory failure.

## Case Report

A18 year old male presented to the emergency department with alleged history of intake of some insecticidal poisonous substance. While in the emergency room (ER) he complained of difficulty in breathing.

Patient's vital signs were as follows: **blood pressure** 135/78 mm Hg,**pulse rate** 120/min, **respiratory rate** 18 breaths/min, and **temperature** 98.6 °C. On chest auscultation there were bilateral basal crackles. The abdominal examination was normal. His pupils were bilaterally pin point with initial neurological examination normal and no evidence of focal neurological deficit.

Laboratory values were normal except an increased white blood cell count (21,000/$cm^3$) with neutrophils predominating. Arterial blood gases on room air were as follows: pH 7.39, $Pco_2$ 36 mm Hg, $Po_2$ 99 mm Hg. Within next two hours he had an sudden episode of desaturation. He was immediately intubated and mechanically ventilated. After intubation he became hypotensive, required vasopressors for three to four hours. Inj atropine drip was started as there was suspicion of organophosphate poisoning. His haemodynamic measures improved with treatment.

Next day arterial blood gases did not show any evidence of an Alveolar-arterial (A/a) gradient difference. In spite of normal A/a gradient the patient failed to extubate even on repeated attempts. This led us to believe that he might have some other cause of respiratory failure, either cardiac or neuromuscular. At this point a more detailed history was obtained from him and his family. It was transpired that prior to hospitalisation he had fever, generalised weakness, right flank pain and loose motion for three to four days. Neurological evaluation was done, considering the possibility ofneuromuscular weakness. Toxicological samples were found out to be negative, on considering this atropine was discontinued.

The neurological examination revealed bilateral weakness of the orbicularis oculi, deltoids, biceps, triceps, interossei, ankle dorsiflexors, and plantar flexors. Rest of the neurological examination including sensory examination was normal. The rapid shallow breathing index was 77. These findings suggested respiratory muscle weakness.

A diagnosis of myasthenia gravis was made on the basis of the history and examination. Neurophysiological studies and acetylcholine receptor antibodies were planned for confirmation. Electromyography studies showed significant decrement on repetitive nerve stimulation, consistent with the diagnosis of myasthenia gravis most probably precipitated by infection.

The patient was started on intravenous immunoglobulin along with intravenous steroids. With the first day intravenous immunoglobulin treatment, his vital capacity improved from 500 ml to 1.4 lit. After next two dayshe passed the weaning trial and was successfully extubated. He received a total of2 gm/kg,i/vIg over five days and continued improvement of muscle strength was found during the rest of his stay in hospital. The acetylcholine receptor antibody panel was positive for binding, blocking, and modulating antibody. Patient was discharged aftertwo weeks on steroids (tapering dose)and pyridostigmine.

## Discussion

As patient presented in ER with alleged history of intake of poisonous substance with signs & symptoms of OP poisoning later on found to be a case of myasthenia gravis presented as isolated acute respiratory failure.

MG is an autoimmune disorder. In about two thirds of patients, extrinsic ocular muscle abnormalities or bulbar weakness may be the. The symptoms usually progress to include the limb muscles [1]. Respiratory failure can be a complication during the late course of MG in about 3 to 8% of cases, known as a myasthenic crisis [2].

Recently, several cases of MG with respiratory failure as a first manifestation have been reported [3-6]. In these studies, respiratory failure as an initial symptom was observed in 14 to 18% of the patients.

As, our patient showed no other neurological symptoms associated with MG, therefore, we did not suspect MG initially. We tried to determine the cause of the respiratory failure. We did not find any evidence of a hypoxemic respiratory failure; as chest X-ray, CT chest and echocardiography was with in normal limit. Therefore, we suspected an acute ventilatory failure.

Based on these results, we investigated the possibility of a neuromuscular disease, especially Guillain-Barre syndrome and myasthenia gravis, the most common and the second most common cause of neuromuscular disease, respectively. However, the cerebrospinal fluid was normal, and the neurophysiological studies showed evidence of myasthenia gravis.In MR crisis remission induction is usually accomplished through the use of high-dose corticosteroids, frequently in conjunction with IV immunoglobulin or plasmapheresis. Maintenance of the remission is usually accomplished by slow tapering of the corticosteroids along with the use of “steroid-sparing” agents, which include azathioprine, thymectomy, and possibly mycophenolatewith anticholinesterase inhibitor.

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