



BMH Medical Journal 2016;3(1):5-9 **Case Report**

A Case Of Unexplained Dyspnoea

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Case Report

50 year old female, without any history of significant illness in the past, presented to our emergency department with episodic shortness of breath and generalized weakness of 3 weeks duration, more towards end of the day. She attributes that her symptoms developed following some mental stress. Over 3 weeks she had many emergency visits and was treated as asthma and anxiety disorder. She was even put on psychiatry drugs.

On examination, she was anxious and tachypneic, with a respiratory rate of 32/min, pulse rate of 86/min, blood pressure of 120/70 mm of Hg and an oxygen saturation of 98%. Respiratory and cardiovascular examinations were normal. But her speech was slurred. On further enquiry, she also gave history of difficulty in swallowing, nasal regurgitation and slurring of speech for last 2 weeks. ENT evaluation was normal. Considering her bulbar symptoms, we made a detailed examination of central nervous system. She had dysarthria, nasal quality of voice and fatigability of speech with no other neurological deficits. All superficial and deep reflexes were preserved. All her routine investigations including chest X-ray (**Figure 1**), ECG, 2D Echo and ABG were normal. Pulmonary function test showed restrictive pattern with maximum inspiratory pressure (MIP) of 33cm H₂O (57% predicted) and maximum expiratory pressure (MEP) of 16cm H₂O (20% predicted) (**Figure 2**). Maximum voluntary ventilation (MVV) was only 49% of predicted. (**Figure 3**).

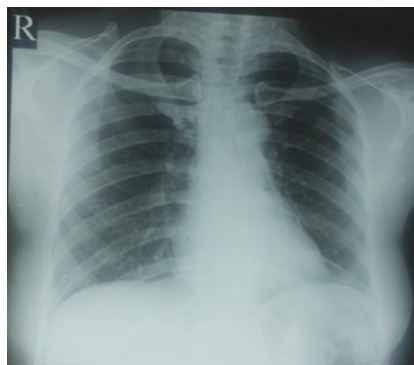


Figure 1: X-ray Chest PA view

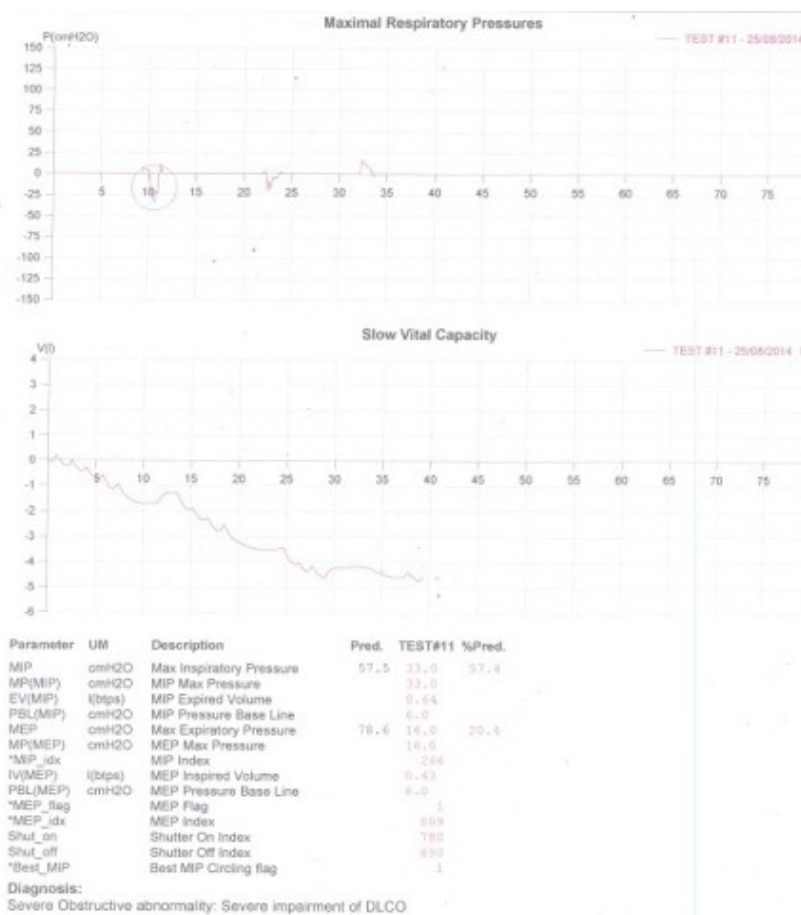


Figure 2: Maximum respiratory pressure

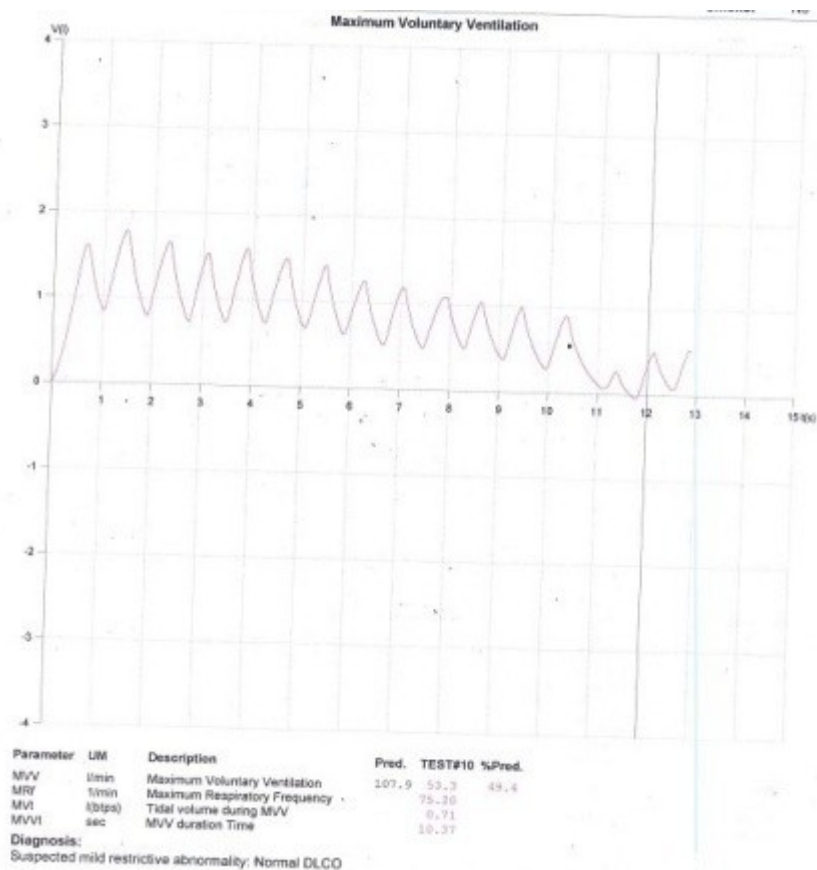


Figure 3: Maximum voluntary ventilation

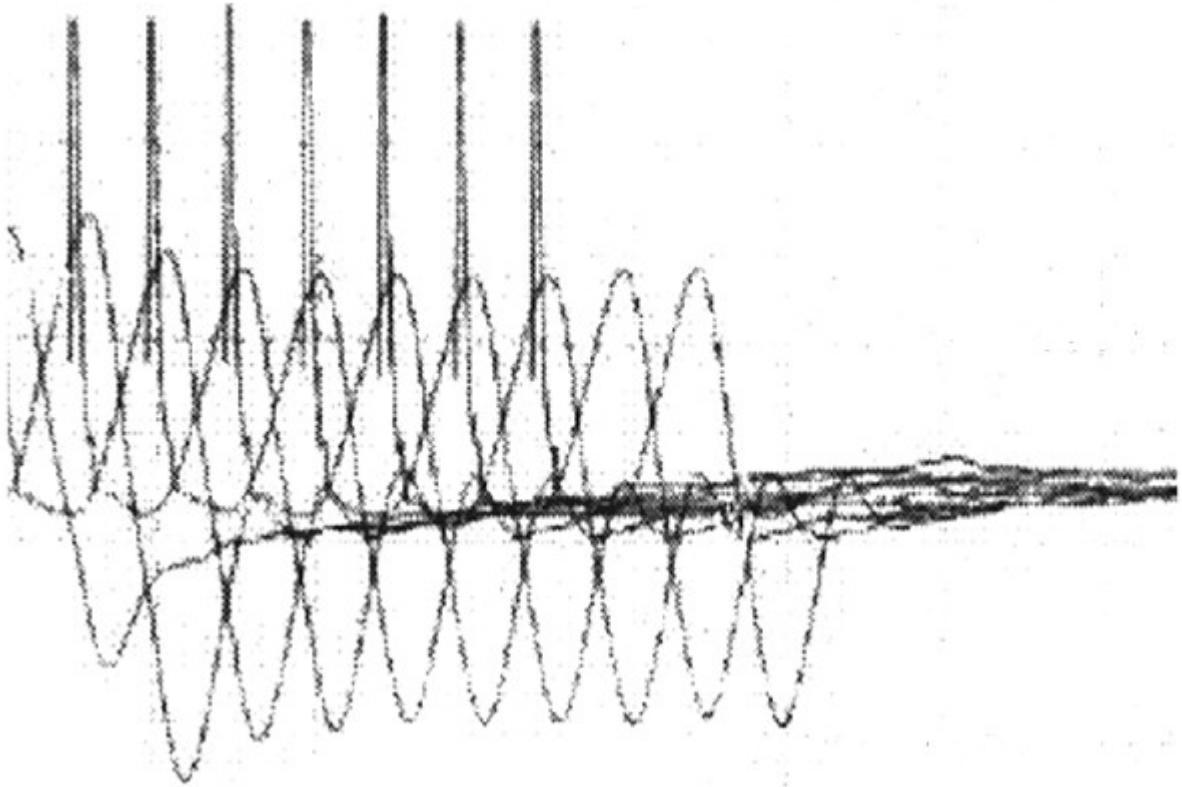


Figure 4: Repetitive nerve stimulation study on left median nerve showing positive decremental response

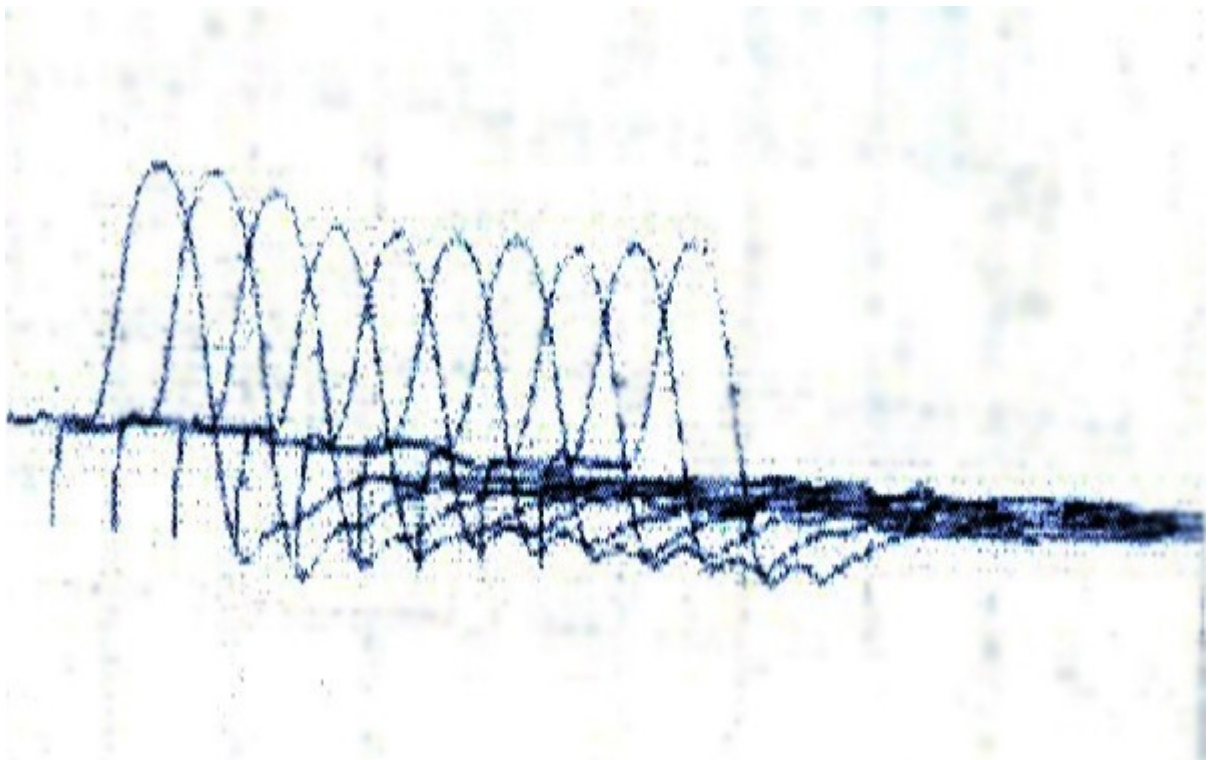


Figure 5: Repetitive nerve stimulation study on right median nerve showing positive decremental response

As she had both respiratory and bulbar symptoms, we suspected a neuromuscular cause for her symptoms. Neurology consultation was done. Nerve conduction study was done as advised by neurologist, which showed significant decremental response on repetitive stimulation (**Figure 4 and 5**). EMG did not show any myopathic changes. She was also confirmed to have antibody against cholinergic receptor. Thus diagnosis of myasthenia gravis was confirmed. CT thorax did not show any thymoma or thymus enlargement.

She was put on pyridostigmine 30 mg TID and prednisolone 5mg TID and she improved dramatically. She was advised to take pneumococcal and influenza vaccination, as she is on long term corticosteroid therapy. She is at present asymptomatic with treatment and is under our regular follow up.

Discussion

Myasthenia gravis [MG] is a neuromuscular disorder of autoimmune origin with antibodies directed against proteins in the postsynaptic membrane of the neuromuscular junction. The prevalence of MG is estimated to be about 150 to 200 per million, with female predominance [1]. MG has a bimodal distribution with an early peak in the second to third decade and a late peak in the sixth to eighth decade.

The hallmark of the disorder is a fluctuating degree of weakness in ocular (50%), bulbar (15%), limb, and respiratory muscles [2]. Weakness worsen towards evening, or after exercise. About 15 percent of patients present with bulbar symptoms like dysarthria, dysphagia, and fatigable chewing. Though isolated respiratory muscle weakness is a less common presentation, involvement of the muscles of respiration can lead to respiratory insufficiency [3]. Acute respiratory failure usually occurs in life threatening "myasthenic crisis", which is usually precipitated by a variety of factors including surgery, stress, infections, medications or tapering of immuno suppression.

Physical findings can vary in myasthenia as the muscle weakness tends to be more when the muscles are stressed. Muscle strength improves with rest. The most distinguishing feature is that sensation and reflexes are preserved in MG. Its confirmed using serologic tests for autoantibodies : *acetylcholine receptors (AChRAb) and/or receptor associated proteins, muscle specific tyrosine kinase (MuSKAb)* and electro physiologic studies (repetitive nerve stimulation studies and single fiber EMG). The repetitive nerve stimulation test, is considered positive if there is decremental response of 15% and has a diagnostic sensitivity of about 75 percent in MG [4]. Acetylcholine receptor antibody is positive in 80-90% of patients with generalized myasthenia gravis [5]. Chest CT or MRI scan should be performed to rule out associated thymoma in all MG patients. In case of respiratory muscle involvement, pulmonary function study reveals mild reduction in vital capacity and moderate reduction in both maximum inspiratory and expiratory pressure.

The treatment of myasthenia includes anti cholinesterase agents, high dose corticosteroids, thymectomy and plasmapheresis in patients refractory to steroid or immunosuppressive therapy. Patients in respiratory failure may require ventilatory support. Patients with generalized myasthenia who develop respiratory infections are at increased risk of myasthenic exacerbations. So current guidelines recommend inactivated pneumococcal and influenza vaccines for these patients.

Conclusion

For any patient presenting with unexplained dyspnoea, we should also consider the rare possibility like myasthenia gravis. Work up for other causes can be exhaustive, expensive, and hazardous if a high index of suspicion for neuromuscular causes is not maintained.

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