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A RARE CAUSE OF BREATHLESSNESS.

General Medicine

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KEYWORDS

INTRODUCTION -
Myasthenia gravis is an established autoimmune disease commonly affecting middle aged female populations . Mediated by a type-II antibody reaction in which antibodies directed against post-synaptic nicotinic acetylcholine receptors attack the myoneural junction and damage the post-synaptic membrane via complement fixation. This results in the failure of action potential propagation across the neurons, eventually leading to a neuromuscular weakness .Classically, the anticholinergic autoantibodies target the extraocular muscles, leading to fluctuating muscular fatigability, predominantly resulting in bilateral diplopia and ptosis, which is typically worse at the end of the day. Breathlessness as a result of diaphragmatic palsy has been rarely described as the initial complaint.

Case report/ Observation –
A 77year old male patient known diabetic on treatment presented to us with breathlessness and tiredness since 2 weeks.

On Day1 of examination, vitals were stable except for SPO2 -80% on RA , 94 % on 6L O2 and RR was 33cpm ,

Respiratory System Examination
Inspection – trachea appears to be central , apex beat not seen,

Palpation – trachea is central , apex beat is medial to MCL ,respiratory movements reduced in Infra mammary , Infra axillary , Infra scapular areas on both sides

Percussion – Impaired note heard over lower lobes in Infra mammary , infra axillary , Infra scapular areas on both sides

Auscultation - Bilateral air entry present , Bilateral NVBS present, absent breath sounds in Infra mammary , infra axillary , Infra scapular areas bilaterally. No added sounds RS examination revealed bilateral lower lobe collapse of lungs .

Basic investigations on day 1:

Blood Investigations	Results	Normal range
Hemoglobin	13.0g/dL	13.5–18.5 g/dL
Platelets	320×10^9/L	150–400×10^9/L
White blood cells Neutrophils	10.2×10^9/L 73%	4.0–11.0×10^9/L 40-75 %
Albumin	32 g/L	35–50 g/L
Alkaline phosphatase	68 U/L	50–150 U/L
Alanine transaminase	77 U/L	5–35 U/L
Random blood sugar	190mg/dl	<200 mg/dl
Creatinine	1.35 mg/dl	0.5-1.11 mg/dL
Sodium	142mEq/L	135–150mEq/L
Potassium	3.19 mEq/L	3.5–5.0 mEq/L
Urea	49.2	10-50 mg/dL
Calcium	8.6 gm/dl	8.5 -11 mg/dL
ESR	7 mm/hr	
C-Reactive protein	6.01 mg/L	<5 mg/L
Blood culture and sensitivity	No organism growth	

Urine routine – normal
TSH– 0.47 µIU/ml
Serology – Non reactive
ECG – showed normal sinus rhythm ,
2D ECHO showed no RWMA, concentric LVH, PASP – 25 mmHg, LVEF – 58%
ABG – Type 2 respiratory failure
USG abdomen and pelvis showed grade 2 fatty liver.

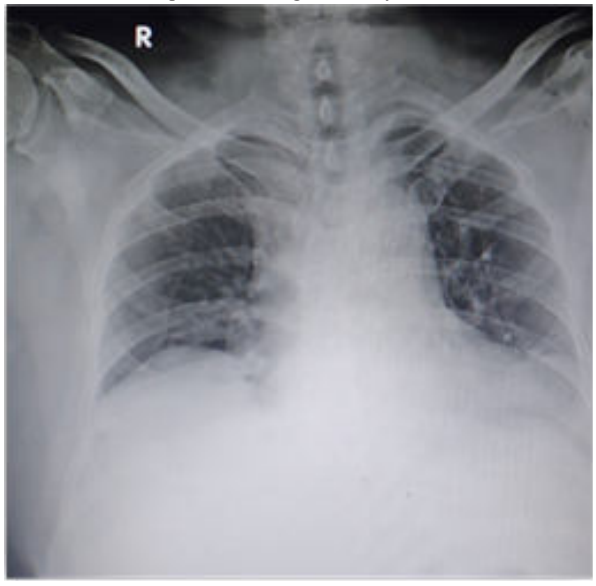

Figure 1: *chest x-ray A/P view*

Chest x-ray could not add much to our clinical findings so we ordered a CT thorax

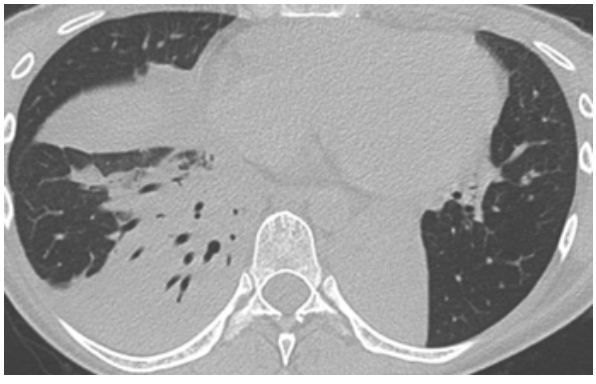

Figure 2 *CT Thorax showed segmental collapse of basal segments of both right and left lower lobes*

On Day 3 Of Examination ,
Patient developed bilateral ptosis of eyelids, we did several

Bedside Clinical Tests –
- Ice pack test

- Rapid Eye blinking test
- Sustained up gaze test
- Single breath count test

All of them were positive !

- Acetylcholine receptor antibodies were negative - 0.27 nmol/L ,
- Anti Musk antibodies were positive

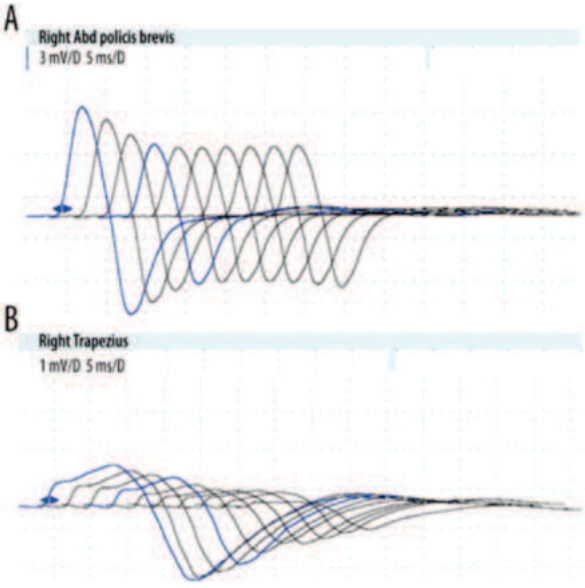

Figure 3 A *Repetitive nerve stimulation testing showed a decremental response*

Usg thorax showed
• Diaphragmatic paralysis on both the sides
• No evidence of thymoma

Course In The Hospital

The symptoms with which our patient presented to us and our clinical , radiological and laboratory findings we came at a diagnosis of Myasthenia gravis involving diaphragm.

Patient was in respiratory distress required intubation, mechanical ventilation and ventilator support .Empirical intravenous antibiotics were initiated. After neurologist consultation plasmapheresis (PLEX) was initiated. In collaboration with nephrologist 6 cycles of PLEX were completed. After significant clinical improvement patient was extubated and transitioned to oral pyridostigmine. Patient was subsequently stabilized and shifted to ward where further clinical improvement was observed.

Oral immunosuppressants and steroids were introduced as part of long term management plan. Oxygen support was gradually tapered and eventually stopped.

The patient was mobilized and after achieving stable clinical improvement was discharged with advice to follow up with the neurologist for continued care and management of myasthenia gravis.

DISCUSSION

Myasthenia gravis (MG) belongs to a spectrum of autoimmune diseases in which anti-acetylcholine receptor antibodies damage neuromuscular junctions.

It is a relatively rare disease with a higher incidence among the female population.[1]

The classical presentation is fatigable fluctuating diplopia or ptosis and, uncommonly, dysphagia or dysphonia. Even though it is rare, this condition can affect any skeletal muscle groups. There have been no reported cases of MG presenting as isolated bilateral lower lobe collapse of lungs secondary to involvement of diaphragm bilaterally. The case described above is an extremely rare presentation of a rare condition, even though myasthenia gravis is the most common neuromuscular junction disorder.

This case highlights the importance of a high clinical index of suspicion for myasthenia in elderly patients, with unexplainable breathlessness even when there is no classical fluctuating weakness.Weakness in myasthenia is due to impaired action potential propagation caused by damage to post-synaptic acetylcholine receptors due to which muscles do not depolarize. The annual incidence of MG is relatively less, often 10-20 newly reported cases per million [2].

The disease follows a bimodal pattern of distribution, with a peak in the second to third decades among the female population and another peak in the fourth to eighth decades among the male population [3]

Significant delays in diagnoses or frequent misdiagnoses among elderly patients have been reported [4].

Owing to a vast amount of possible causes for neuromuscular symptoms,

- Transient ischemic attack (TIA) or stroke
- Parkinson's disease
- Motor neuron disease
- Neuropathies
- Horner syndrome,

myasthenia is thought to be under-diagnosed in senile patients [5].

Treatment Options

There are 3 principal treatments for myasthenia gravis:
1. Acetylcholinesterase inhibitors
2. long-term immunosuppressive agents
3. Rapid immunomodulatory therapy

In patients with thymomas, a thymectomy is considered as a treatment option.

CONCLUSION

There have been no reported cases of myasthenia gravis presenting only with diaphraghmatic palsy manifesting as breathlessness as there first symptom in the elderly male population. The elderly are at risk of misdiagnosis, leading to further complication. This case shows that myasthenia should be under consideration as a possibility in elderly patients presenting with breathlessness and tiredness despite the absence of classical symptoms. It is important to diagnose myasthenia in its early stages and manage it appropriately.

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