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Non-resolving pneumonia with respiratory failure: scratch your neurons too

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Abstract

Finding the etiology for non-resolving or recurrent pneumonia is enough to cause physicians to have sleepless nights. Moreover, if the reason turns out to be the primary presentation of neuromuscular disease, it takes all of us aback. The differential diagnosis should include atypical infections, malignancy, and neuromuscular disorder. We present a case of a 35-year-old female who presented with non-resolving pneumonia with respiratory failure but was later diagnosed with myasthenia gravis.

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Introduction

Pneumonia refers to infection of lung parenchyma due to varying etiology. Usually, these cases resolve spontaneously or after a short course of antimicrobials. In some cases, the extended use of antimicrobials can be required especially in patients with considerable comorbidities or with immunodeficiency. In a few patients, the clinical symptoms as well as radiological opacities persist even after prolonged use of antimicrobials as per protocol. Some studies consider that at least 4-6 weeks should be considered before labeling these patients as non-resolving pneumonia.¹

Neuromuscular weakness is a known but rare etiology leading to pneumonia. Myasthenia gravis, spinal muscular atrophy, Duchenne muscular dystrophy, and Pompe Disease are some known neuromuscular disorders that can lead to recurrent pneumonia.²

Patients suffering from myasthenia gravis usually present with ptosis, dysphagia, dysarthria, limb muscle weakness, and respiratory muscle paralysis.³ This leads to micro aspiration and patients present with pneumonia and respiratory failure at later stages of the disease. We present a case of a 35-year-old female who presented with non-resolving pneumonia with respiratory failure but was later diagnosed with myasthenia gravis. However, the latter presented as dysphagia leading to aspiration pneumonia and respiratory failure as the primary presentation which adds to the rarity of this case.^{4,5}

Case Report



A 35-year-old non-smoker, immunocompetent female was referred to our Himalayan Institute of Medical Sciences, Swami Rama Himalayan University in an intubated state. She had complaints of dyspnea of grade 4 according to the modified Medical Research Council), fever, and cough with expectoration for 6 months which had progressed 3 days back for which she was intubated. She had a high oxygen requirement of FiO₂ as high as 80%. The rest of her systemic examination was normal. She had a high total leukocyte count (23,000 cells/cumm). Chest roentgenogram revealed left lower zone consolidation (Figure 1).

Contrast-enhanced tomogram thorax showed segmental collapse of medial and posterior basal segments of the left lower lobe with few fibrotic lesions in lingular segments of the left upper lobe suggestive of infective bronchopneumonia (Figure 2). The endotracheal culture was inconclusive. With suspicion of any endobronchial lesion, a bronchoscopy was done; however, no abnormality was found. Bronchoalveolar lavage was sent which showed growth of Pseudomonas. She was started on piperacillin tazobactum based on her sensitivity. She responded clinically and she was weaned off as per protocol and extubated. But soon after extubation, patient redeveloped respiratory distress with increased oxygen demand. She was re-intubated in view of increasing respiratory distress. History was revisited and it was found that she had off-and-on complaints of difficulty in swallowing since the last year, hence, we thought of two differentials neuromuscular diseases and motor neuron diseases.

As the patient had no history of muscular weakness, wasting of muscles, slurred speech, bladder or bowel incontinence; motor neuron disease was unlikely. Neuromuscular diseases were still

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thought of. Serum acetylcholine receptor binding antibody was found to be >8.00 nmol/L (normal <0.40). Functional upper gastrointestinal endoscopy was done which showed diminished swallowing reflex. The same was repeated after the instillation of 0.5 mg neostigmine and the swallowing reflex showed marked improvement (Figure 3). The patient was diagnosed as myasthenia gravis of oropharyngeal muscles which led to aspiration pneumonia and weakness in

the respiratory muscle.

The patient was tracheostomized and started on neostigmine and corticosteroids after due consultation with a neurologist. She was weaned off of the ventilator. After 2 weeks, there was marked clinico- radiological improvement. She was discharged with Ryle's tube and tracheostomy tube *in situ*. At follow-up, after 8 weeks, her pneumonia has completely recovered and she is under neurological care.

Discussion

Non-resolving pneumonias are those pneumonias that have an unrecognized etiology. Unless we discover that etiology and treat it, it is impossible to cure the patient of his disease. Delay in diagnosis can lead to mortality too. Incorrect diagnosis, inadequate antibiotic therapy, impaired host defense, atypical organisms, resistant pathogens, non-infectious causes, tuberculosis, endobronchial lesions, etc. are the common causes of non-resolving pneumonia or slowly resolving pneumonia.^{6,7}

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Neuromuscular diseases leading to non-resolving pneumonia are a very rare entity. Hence initially,

there should be a step ladder pattern to exclude common causes first. In our case too, we first tried

to find out causes like atypical organisms and did a bronchoscopy to find any endobronchial cause.

We treated the Pseudomonas with sensitive antibiotics. The reason that she had an extubation

failure despite any known comorbidity or electrolyte imbalance, made us think for an alternate

diagnosis.

Myasthenia gravis is an autoimmune disease where autoantibodies target the acetylcholine

receptors at the presynaptic levels. In myasthenia gravis impaired cough mechanism, inspiratory,

expiratory, and bulbar muscle weakness make it difficult for secretions in the lower airway to clear

up.8 This can lead to chronic aspiration as was seen in our case. In a study by Stephan et al., Gram-

negative non-fermenter bacilli were shown to be the most common cause of pneumonia in patients

with myasthenia. In our patient too, *Pseudomonas aeruginosa* was traced. Selection of antibiotics

should be preferably done under the umbrella of culture and sensitivity whenever possible.¹⁰

Non-resolving and recurrent pneumonia in patients with myasthenia gravis leads to a significant

increase in morbidity and mortality. Hence early identification of the same and tailored treatment

is essential.

Conclusions

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The key to treating any disease is to understand the etiology, then only appropriate targeted therapy can be provided to the patient. As in our patient, though pneumonia was treated until the treatment for myasthenia was given to the patient, our treatment was not complete.



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Figure 1. Chest X-ray revealed left lower zone consolidation.



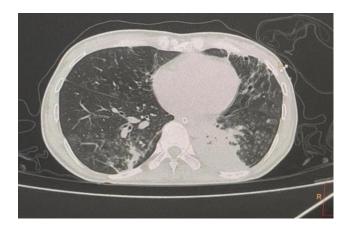


Figure 2. Contrast-Enhanced Computed Tomography (CECT) of the thorax showed segmental collapse of medial and posterior basal segments of the left lower lobe with few fibrotic lesions in lingular segments of the left upper lobe.





Figure 3. Upper Gastrointestinal (GI) endoscopic image showing normal swallowing after neostigmine.





Figure 4. Chest X-ray showing improvement in consolidation with tracheostomy in situ.