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Case Report: Dysphagia in Inclusion Body Myositis Leading to Respiratory and Gastrointestinal Complications

Veroneka Mikhail  
*Jefferson Health NJ*

James A. Espinosa  
*Jefferson Health NJ*

Alan Lucerna  
*Jefferson Health NJ*

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Inclusion body Myositis (IBM) stands as a rare and complex neuromuscular disorder (NMD) characterized by progressive muscle weakness and atrophy. Among its cardinal symptoms are dysphagia and respiratory distress, which are the most common cause of death in this disease. While the differential diagnoses of respiratory distress is vast and includes aspiration, pneumonia, acute coronary syndrome, emphysema, and congestive heart failure, a clinician should recognize that respiratory distress can also be secondary to dysphagia in NMDs like IBM and can quickly become life threatening. Here we present the case of a 68-year-old female with a history of IBM who presented for respiratory distress, was found to have severe dysphagia, and subsequently required intubation and percutaneous endoscopic gastrostomy (PEG) tube placement.

**Case Presentation:**
68-year-old female with a history of inclusion body myositis presented to the emergency department (ED) in respiratory distress with chief concern of sudden shortness of breath that occurred prior to arrival after she aspired while eating cheese and crackers, and drinking wine. At that time, patient (pt) denied any chest pain, coughing, nausea, and vomiting. Pt explained that approximately six years prior to this visit she underwent series of tests including multiple barium swallow studies, electromyography, and anti-nuclear antibody (ANA) testing and was diagnosed with IBM. She also explained that she had multiple hospitalizations in the past due to aspiration pneumonia and even a tracheostomy in 2019.

Upon arrival to the ED, pt’s vitals were as follows: Heart rate 117 beats per min, respiratory rate 30 breath per minute, oxygen saturation (O2) 82%, blood pressure 162/75 mmHg, and temperature 99.2 degrees F. The physical exam showed mild tachypnea, intercostal retractions, bilateral inspiratory wheezing, and diminished breath sounds at the right lung base. Lactate 2.6, no leukocytosis. All other labs within normal limits. Electrocardiogram showed sinus arrhythmia 99 beats per minute, no ST changes, no QT prolongation. Pt was immediately placed on 6L nasal cannula (NC) with minimal improvement in O2 status, then transitioned to 11L oxygen with O2 saturation 96%. Arterial blood gas showed pH 7.44, pCO2 40, pCO2 81, bicarb 27. Chest x-ray (CXR) showed lobar aspiration pneumonia with mild peribronchial interstitial edema. Computerized tomography angiogram (CTA) scan performed and showed mild enlarged pulmonary trunk, and patchy airspace opacities in the right upper, right lower, and left lower lobes representing lobar aspiration pneumonia (figure 1). Pt was given antibiotics (doxycycline and ceftriaxone), dexamethasone, tapering dose of dexamethasone, placed on nothing by mouth (NPO), code status confirmed as full code, and pt was admitted to the intensive care unit for sepsis and acute hypoxic respiratory failure secondary due to multi-lobar aspiration pneumonia.

In the ICU, nasogastric tube (NGT) was placed, pt was started on triclel feedings at 20mL/hr and O2 saturation stabilized and pt was weaned down to 6NC. Overnight, pt de-saturated to 80%, was titrated on NIPPV, and oxygen was further increased, then placed on 50L at 50% high flow nasal cannula (HFNC) and continued to show signs of respiratory distress including O2 stat “80%”, increased work of breathing, copious oral secretions, and bilateral wheezing despite multiple suctioning. Pt was subsequently intubated, Infectious disease consulted, and she was swabbed to evacuate the airway. Pt was successfully weaned from the ventilator over the next 3 days. Pt’s shortness of breath resolved and HFNC and intubation were gradually weaned down to 2L NC and wanted NGT removed. Pt was kept NPO, incentive spirometer, aspiration precautions, head of bed elevated, Physical therapy, Occupational therapy, and Speech and Swallow therapy were placed. Speech evaluation performed via Videofluoroscopic swallow studies showed severe pharyngeal dysphagia, pooling of food consistencies, and moderate esophageal dysphagia (figure 2). It was determined that pt is unable to tolerate by mouth (PO) diet and is a high aspiration risk. Pt declined NGT, dobbuff (DHT), and PEG despite multiple detailed discussion and wanted to have nutrition PO as she did at home. The patient’s family gave her Jelco PO against medical advice, and soon after pt developed another episode of respiratory distress where she became hypoxic SpO2 86-89% on HFNC 100% 5HIQ 331pm. Stat portable CXR shows complete atelectasis of the left side with suspected mucus plugging (figure 3). Pt was agreeable to intubation and immediate temporary DHT placement, and eventual a PEG tube due to malnutrition in the setting of recurrent aspiration. Spumum cultures grew pseudomonas and pt was started on 7 days course of meropenem, 3% saline nebs, and chest physical therapy. Gastroenterology was consulted and PEG tube was placed once pt’s respiratory status stabilized. Pt was extubated 4 days after PEG tube placement, tube feedings were up-titrated slowly until goal of 41mL/hr was met. Pt continued to clinically improve and was stable for discharge home 1 week later.

**References:**