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Selected Topics: Neurological Emergencies

EMERGENCY DEPARTMENT MANAGEMENT OF A MYASTHENIA GRAVIS PATIENT WITH COMMUNITY-ACQUIRED PNEUMONIA: DOES INITIAL ANTIBIOTIC CHOICE LEAD TO CURE OR CRISIS?

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□ **Abstract—Background: Myasthenic crisis is a rare, yet serious condition that carries a 3%–8% mortality rate. Although infection is a common cause of decompensation in myasthenia gravis, several antibiotics classes have also been associated with an exacerbation. Selecting antibiotics can be a daunting clinical task and, if chosen inappropriately, can carry significant deleterious consequences. Not only do clinicians have to focus on treating the underlying infection appropriately, but avoiding antibiotics that may potentiate a myasthenic crisis is also vital. Case Report: An 85-year-old female with a history of myasthenia gravis presented to the emergency department (ED) with increasing generalized weakness and shortness of breath. Clinical work-up was consistent with a community-acquired pneumonia (CAP) diagnosis. Her medical history included a myasthenia gravis exacerbation shortly after receiving moxifloxacin for CAP. After reviewing the patient's allergies, as well as potential antibiotic triggers, the decision was made to treat with tigecycline. The patient responded well to tigecycline therapy and was deemed stable for discharge on day 4 of hospitalization. Why Should an Emergency Physician Be Aware of This?: Evaluation of the myasthenia gravis patient frequently originates in the ED. It is important for clinicians to be able to distinguish between an underlying illness and a myasthenic crisis. In the event of an infectious process causing clinical deterioration in a myasthenia patient, optimal antibiotic selection becomes paramount. This patient case highlights the addition of tigecycline to the armamentarium of therapies available to treat myasthenia gravis patients presenting to the emergency department with CAP.** © 2016 Elsevier Inc.

□ **Keywords—myasthenia gravis; community-acquired pneumonia; tigecycline; myasthenic crisis; infection**

INTRODUCTION

Myasthenia gravis is an autoimmune disorder affecting 5–15 people per 100,000 (1). A myasthenic crisis occurs when antibodies are formed against nicotinic acetylcholine receptors, causing the inability to sustain a neuromuscular contraction, and is often defined as acute respiratory failure prompting intubation. Although many patients with myasthenia are controlled with acetylcholinesterase inhibitor medications, acute myasthenic crisis affects 15%–20% of patients with myasthenia and carries a 3%–8% mortality rate (1). A myasthenic crisis can be precipitated by a number of factors; however, infection is responsible for 40%–70% of crisis episodes (1,2). Additionally, several medications, including antibiotics, have been implicated in causing a myasthenic crisis. The unique situation of treating an underlying infection without precipitating or worsening

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a myasthenia crisis can be difficult to accomplish, especially in a patient with medication allergies. We present a case of success using tigecycline for treatment of community-acquired pneumonia (CAP) in a patient with myasthenia gravis and to provide a brief overview of other antibiotics associated with the development of myasthenic crises.

CASE REPORT

An 85-year-old female presented to the emergency department (ED) with a 1-week history of shortness of breath, cough, increasing generalized weakness, and decreased appetite. The family of the patient reported seeing increased work for breathing and inability to expectorate. She was still able to complete activities of daily living and did not complain of any arthralgias, myalgias, or chills. Her medical history included myasthenia gravis for 18 years; controlled, treated hypertension; atrial fibrillation with a rapid ventricular response, controlled and in normal sinus rhythm throughout hospitalization; congestive heart failure with an estimated ejection fraction of 35%−40% 6 months prior; pacemaker placement; right breast cancer diagnosed 7 months prior, undergoing treatment; colon cancer status post resection 14 years prior; and chronic hearing loss. The patient reported an allergy to penicillin, but was unable to recall the previous reaction to the medication. Per the patient's report, there was a family history of severe anaphylactic reactions to penicillins. Our medical records did not reveal a previous administration of a penicillin or cephalosporin. Her home medications included pyridostigmine 180 mg orally (p.o.) daily, azathioprine 50 mg p.o. daily, prednisone 7.5 mg p.o. every other day, rivaroxaban 15 mg p.o. daily, digoxin 0.25 mg p.o. daily, aspirin 81 mg p.o. daily, omeprazole 40 mg p.o. daily, lisinopril 2.5 mg p.o. daily, carvedilol 3.125 mg twice daily, furosemide 20 mg p.o. daily, exemestine 25 mg p.o. daily, and calcium carbonate 200 mg p.o. daily.

Upon physical examination, the patient was noted to be mildly tachypnic at a respiratory rate of 22 breaths/min with some use of accessory muscles, but was able to speak in full sentences. Heart rate and blood pressure were stable at 80 beats/min and 132/55 mm Hg, and the patient had an oral temperature of 39.3°C. Her initial oxygen saturation was 96% on room air, which increased to 100% on 2 L oxygen delivered via nasal cannula. On auscultation, breath sounds were clear bilaterally without wheezes, rales or rhonchi appreciated. A negative inspiratory force was obtained and felt to be adequate for the patient to proceed without ventilator support (value not documented). She had a 4 out of 5 strength in bilateral upper and lower extremities and was able to do repetitive motion without tiring. Her cranial nerves and sensation were noted to be fully intact. Admission laboratory values included a basic metabolic panel within normal limits, pro−brain natriuretic peptide of 1394 pg/mL (normal range 0−125 pg/mL), and a white blood cell count of 10.7 thousand cells/μL with 9% bands. Blood cultures obtained on admission were negative for growth, and a urinalysis was not performed.

A chest x-ray study was initially obtained and was interpreted by radiology as cardiomegaly with central vascular congestion, an elevated left hemi-diaphragm and trace pleural effusion in the left lung base. Given these findings and an overall lack of clarity for a clinical diagnosis, a chest computed tomography was obtained showing "bibasilar airspace disease and consolidation, pneumonia and atelectasis are primary differential considerations," and the decision was made to initiate antibiotics for CAP. Tigecycline 100 mg i.v. bolus was administered, followed by 50 mg every 12 h until the patient was discharged on doxycycline. The patient responded well to antibiotics and was stable for discharge after 3.5 days.

It is notable that 6 months before this hospitalization, the patient was admitted for a similar shortness of breath episode due to myasthenia gravis and CAP. She presented in a similar fashion with progressive worsening of shortness of breath and wheezing. The diagnostic differential included both myasthenia gravis and CAP, and the patient had initial doses of aztreonam, azithromycin, and moxifloxacin (two doses). Approximately 16 h after the second administration of moxifoxacin, the patient required intubation and was placed on mechanical ventilation. Additionally, the patient had a diffuse, morbilliform rash involving the chest and abdomen, as well as some of the upper extremities, and an erythematous face. At the advisement of neurology, the patient underwent plasmapheresis and antibiotics were changed to tigecycline. It was not possible to fully determine during chart review whether the intubation was required secondary to worsening myasthenic crisis (due to either infection or medication administration), acute pneumonia, or both. The patient was extubated 7 days later and discharged to a rehabilitation facility on hospital day 16.

DISCUSSION

We report a case of successful use of tigecycline for presumed CAP in a patient with myasthenia gravis and an antibiotic allergy. Given her history of potential antibiotic-induced myasthenic crisis, antibiotic selection in the ED for treatment of CAP (in accordance with current Infectious Disease Society of America recommendations) and avoidance of trigger medications were of crucial importance. Choice of antibiotic therapy in a patient with myasthenia can be challenging, as many