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A Case of COVID-19 Related Myasthenic Crisis: Lessons Learned

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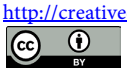

Abstract

Background: COVID-19 was initially described to affect the respiratory system, it is now known to affect other systems and present in a myriad of ways. This has often proved to be a diagnostic and therapeutic challenge to clinicians and stretched healthcare resources, sometimes with poor outcomes. We report on an atypical presentation of SARS-CoV-2 infection as an acute exacerbation of myasthenia gravis in a low resource setting. **Case Presentation:** A middle aged male with myasthenia gravis presented with a three weeks' history of worsening generalized weakness and three days' history of new-onset dysphagia with no history of fever and cough. After a few days of apparent improvement, the patient developed progressive respiratory distress. Further evaluation confirmed bilateral pneumonia and COVID-19. **Conclusion:** There is a need for clinicians and health service providers to have a low threshold for suspicion and testing for COVID-19 in patients with pre-existing medical conditions and immunomodulatory therapy such as Myasthenia gravis.

Keywords

Myasthenia Gravis, COVID-19, SARS-COV-2, Low Resource Settings

1. Background

Coronavirus disease (COVID-19) has impacted the health of individuals and nations all over the world. [1] [2] [3] [4] Though the respiratory system was the main site of infection and inflammation, other organs and systems such as the nervous system have also been shown to be affected [5]. A plethora of neurolog-

ical symptoms have been reported with the most common symptoms being headaches, myalgia, altered mental status, taste and olfactory dysfunctions. They are more common in hospitalized COVID-19 patients with estimates being as high as 73% in one cohort. Central nervous system manifestations were mainly acute cerebrovascular diseases, posterior reversible encephalopathy, post-infectious encephalitis and myelitis. Guillain-Barré syndrome, cranial neuropathies and critical illness myopathy are common manifestations of peripheral nervous system disorders of COVID-19 [6] [7].

These manifestations have been associated with more severe disease, longer hospital stay, higher in-hospital death rate and increased risk of complications, disability and sequelae on discharge [8].

Consequently, SARS-CoV-2 infection is recognized as a chameleon/mimic whose prompt detention and treatment should be the focus for healthcare workers during this pandemic and beyond in order to protect the health of individuals and communities. We report on a case of COVID-19 which presented as an acute exacerbation of generalized myasthenia gravis and lessons learned.

2. Case Presentation

A 47 year old man previously diagnosed with generalized myasthenia gravis presented to the Accidents and Emergency department with a three weeks' history of worsening generalized weakness and three days' history of new-onset dysphagia. There was no history of fever, cough, chest pain, recent travel and contact with COVID exposure. He had no history of recent surgery, immunization, tapering of immunosuppressive medications and intercurrent illness. He however reported receiving unspecified medications at two clinics as well as scarifications and herbal concoctions as alternative traditional healthcare for his symptoms prior to presentation at the hospital. Pre-admission medications were Pyridostigmine 240 mg BID, Prednisolone 15 mg OD and Metformin 500 mg BID.

The initial physical examination revealed a body temperature of 37˚C, oxygen saturation of 96% in ambient air, pulse rate of 74 beats per minute (bpm), blood pressure of 150/90 mmHg and respiratory rate of 18 cycles per minute (cpm). Neurological evaluation showed marked bilateral ptosis, motor power of MRCUK grade 4 and 3 in the upper and lower extremities respectively.

Intravenous fluid was commenced and Pyridostigmine increased to 240 mg TID. Patient's clinical condition remained stable over the next three days but subsequently deteriorated with worsening of respiratory status (respiratory rate of 26 cpm, crepitations over the right lung zone, oxygen saturation of 83% under ambient air, pulse rate of 100 bpm, blood pressure of 210/120 mmHg) necessitating oxygen delivery via intranasal cannula which temporarily improved SPO_2 97% and oral Nifedipine XL 30 mg BID was commenced. However, the patient's desaturation continued (SPO_2 85% on oxygen, tachypnea of 34 cpm). Intensive unit care admission was planned but was deemed infeasible due to lack of a

functional ventilator and referral to a centre with one was planned. IV methylprednisolone, SC regular Insulin and IV Ceftriaxone was commenced while the dose of pyridostigmine was escalated to 240 mg QID and IV Neostigmine was introduced. Complete blood count returned with a packed cell volume of 37%, leucocytosis of 21.9 × 10^9/L (neutrophils 91.3%, lymphocytes 6.2%, monocytes 2.0%, platelets 198 × 10^9/L). Blood chemistry revealed serum potassium 3.61 mmol/L (3.5 - 5.0), sodium 147 mmol/L (135 - 145), chloride 103 mmol/L (90 - 106), urea 14 mg/dL and creatinine 0.6 mg/dL.

On the 9th day on admission, chest computed tomography scan confirmed bilateral pneumonia and COVID screening was ordered but patient died before results were available. Two days after his demise, nasopharyngeal swab PCR returned positive for SARS-COV.

All healthcare workers who had contact with him and other patients on admission in the same ward were screened for SARS-COV-2. Two healthcare workers and two patients' samples returned positive and subsequently made uneventful recoveries.

3. Discussion

This patient was well known to the clinical staff, who had interacted with him on previous visits and were familiar with his clinical history. Thus, his presentation with exacerbation of myasthenic pattern weakness was initially attributed to poor adherence to medications. Additional factors that may have contributed to our low index of suspicion included a lack of travel history at period when community spread was assumed to be a low risk and lack of known exposure to suspected or confirmed COVID-19 cases. Perhaps the most crucial factor that led to diagnostic delay was the absence of fever, cough or difficulty in breathing on admission (all patients seen at the emergency department underwent a screening by a triage nurse). The absence of key symptom indicators may have been due to prior steroid therapy (prednisolone) or may indicate a low sensitivity of these symptoms and its poor performance in general or particularly in individuals with pre-existing health conditions. Ultimately, these factors accounted for the extremely late suspicion and disastrous outcome. Subsequent contact tracing and testing among hospital contacts suggested an intra-hospital replication rate of at least 4 (only potential aerosol generating procedures carried out were intranasal oxygen delivery and oral suctioning). Patient to healthcare worker transmission has been well documented, however, this report also highlights patient-patient spread of SARS-CoV2 in hospitals with open wards and with patients in relatively close proximity.

In-hospital COVID-19 acquisition, although a distinct possibility, was not deemed likely in this case due to 3 week duration of his symptoms, treatment behavior of seeking alternative healthcare resulting in a late presentation to the hospital. Moreover, the occurrence of death on the 10th day of admission was significantly earlier compared to reported median time to COVID-19 death of

18.5 - 21 days. [9] [10] Finally, tachypnea on initial presentation (18 cpm) suggests respiratory manifestation of COVID-19 and indicate a prehospital transmission.

COVID-19 infection with coexisting Myasthenia gravis has been previously reported in developed countries where patients were treated with intravenous immunoglobulin and plasmapheresis. Good outcomes were reported in these series in contrast to this index case. [11] [12] [13] These medication and procedure are scarce and expensive, and as a result, are out of reach for many patients in myasthenic crisis in low resource settings. It is quite likely that recovery from COVID-19 may represent a significant hurdle for myasthenia gravis patients in resource-limited settings such as ours.

4. Conclusion

Low threshold for suspicion and testing for COVID-19 in patients with pre-existing medical conditions and immunomodulatory therapy such as myasthenia gravis who develop acute exacerbation may need to be adopted by health professionals in order to avoid undue morbidity and mortality during and after this current pandemic.

Consent for Publication

The patient died and consent for publication was sought and obtained from a relative.

Authors' Contributions

FMO summarized the case report and wrote the first draft. FMO, AMF, OMF, KMA, OOO and BSO interpreted clinical data and made major contributions to the manuscript. All authors read and approved the final manuscript.

Availability of Data and Materials

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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