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COVID-19: A Family's Cursed

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ABSTRACT

Background: *This report aims to observe a clinical association between obesity, and COVID-19 symptoms and post infection symptoms. Along with the importance of supportive treatment, in detection of COVID-19 complications, as early detection and intervention made a huge difference in patients' prognosis.*

Subjects and Methods: *After verbal consent and clearance from ethics committee, all the 3 patients (n=3) presenting with common cold, fever, cough, and breathlessness, along with obesity were considered for entry to this study. The diagnosis of COVID-19 was confirmed via a positive rtPCR test among all the patients. All the patients were given standard treatment.*

Results: *All the 3 cases were obese adults aged 51 years old male, 33-year-old male, and 45- year-old female, all of them presented with fever, cough, breathlessness, and body ache simulating a respiratory tract viral infection. They were initially treated with Cephalosporin IV 1 g, Levaquin 750 mg daily, and subcutaneous injection of low molecular weight heparin 40 mg BID, Dexamethasone 6 mg IV BID depending upon the clinical status of these patients. Those patients who had lower oxygen saturation below 90% (n=2) were also administered dexamethasone dosage to 8 mg BID IV, and meropenem to 1 g IV BID, and continue supportive treatment for possible benefit. All patients recovered.*

Conclusions: *Over the course of our 3 cases series, we were able to observe a clinical association between obesity, and COVID-19 symptoms, and post-infection symptoms. While this is a preliminary outcome on the impact, it does raise questions about disease modifications in patients with obesity, and its impact on response to treatment. We also saw the importance of supportive treatment, in the detection of COVID-19 complications, as early detection and intervention made a huge difference in patients' prognosis.*

Keywords

COVID-19, Type 2 diabetes, Cardiovascular diseases, Hypertension, Cancer.

Introduction

There is evidence that severe acute respiratory syndrome can result from coronavirus 2 (SARS- CoV-2), which may predispose COVID-19. It may have a spectrum of manifestations especially in obese patients that range from silent infection to serious illness [1]. COVID-19 may be characterized by pneumonia, consolidation, in the lower lung zones that may extend to upper zones and predispose respiratory failure, causing death in about 0.5% of confirmed cases [2]. More than 90% of patients infected with COVID-19 showed mild or no symptoms but the rest of the

cases infected showed severe symptoms resulting in significant mortality. Since COVID-19 spreads rapidly and relentlessly, as a pandemic around the world, we have an unmet need to develop new therapies for this disease. However, age along with patients with underlying conditions like Type 2 diabetes, cardiovascular diseases, hypertension, and cancer have an increased risk of severe disease and death due to COVID-19 infection. Obesity has recently been emerged as a novel risk factor for hospitalization and death due to COVID-19. Several studies have observed that people with obesity are at a higher risk of severe disease and death due to COVID-19 [3]. Currently, there is no evidence-based therapy and use of any treatment, is open to bias as an antiviral agent for COVID-19. The agent dexamethasone is a type of corticosteroid which is a potential anti-inflammatory agent could be protective in serious patients predisposed to respiratory failure. Most viral infections are associated with mild to no adaptive immunity to the virus. The host with COVID-19 may be dependent on innate immunity for inhibition of the severity of disease. The action of interferons may provoke innate response which is considered essential [4]. There are a few key orchestrators of the immune response that may be comprised of an antiviral and other functions characterized with immunomodulation [5]. Further research indicates that type I interferon (interferon-β) which is a potential cytokine induced by virus is known to provoke innate immune responses in the respiratory system [5,6]. It is possible that the release of interferon-β in vitro, is inhibited by SARS-CoV-2 [7]. However, a clinical trial among patients with COVID-19 revealed significant decline in the interferon activity in serious patients [8]. In high-risk subjects above 45 years, having obesity, and among those taking immunosuppressive medication, who generate less interferon-β, may contribute to extensive pulmonary disease [9]. The primary objective of this study is to observe a clinical association between obesity, and COVID-19 symptoms and post infection symptoms. Along with the importance of supportive treatment, in detection of COVID-19 complications, as early detection and intervention could possibly made a huge difference in patients' prognosis.

Case 1

51-year-old obese male patient with a medical history of uncontrolled HPN. He presents with low grade fever, severe joint and muscle pain, and cough. Gradually his condition deteriorated after two days his O_2 levels dropped below 85%, and continues deteriorating to 70% with SOB, nausea, and vomiting. The patient was reluctant to be taken to the hospital; he asked to be treated at home. Cephalosporin IV 1 g, Levaquin 750 mg daily, and subcutaneous injection of low molecular weight heparin 40 mg BID, Dexamethasone 6 mg IV BID. The patient condition continued deteriorating; he became hyperglycemic and was treated with insulin. His antibiotics were changed to meropenem 500 mg 3x, and his Dexamethasone was increased from 6 mg IV BID to 6 mg IV TID. The patient continued deteriorating clinical condition, we used streamed humidified air daily, and he was started on b2 agonist on nebulizer every 4 hours. There was a mild improvement as his O_2 stat reached 89% on O_2. Echo study was normal after two days, the Dexamethasone was increased again to 8 mg IV BID, added Levaquin IV. The patient saw continued improvement, and within 3 days the O2 required consumption dropped from 10 L to 4 L.

Case 2

33-year-old healthy male patient without any past medical history, patient developed cough, mild fever, and joint pain after taking care of his mother who was infected with COVID-19 for 10 straight days. He contacted his family doctor who advised him to isolate himself and take some over the counter fever reducer. After 5 days, the patient condition continued to worsen, where he began hallucinating, and developed severe diarrhea, and from there also developed hypotension. It was at that point that the treating team placed him, supportive IV fluid, plus 6 L of O_2, and 6 mg BID IV dexamethasone, alongside with 500 mg IV TID of meropenem, bronchodilator. The patient condition continued deteriorating despite measure, after 3 more days, the patient O_2 need was increased to 10 L, his CT scan ordered showing bilateral symmetrical fibrosis of lower and upper fields.

The decision was made to increase the dexamethasone dosage to 8 mg BID IV, and meropenem to 1 g IV BID, and continue supportive treatment, after 3 days patient showed first signs of improvement, after 10 days dexamethasone was tapered offer from 10, to 8, to 6, and 2 mg, continued for 2 weeks. We also tapered medications from meropenem, to Levaquin 750 mg for 7 days,

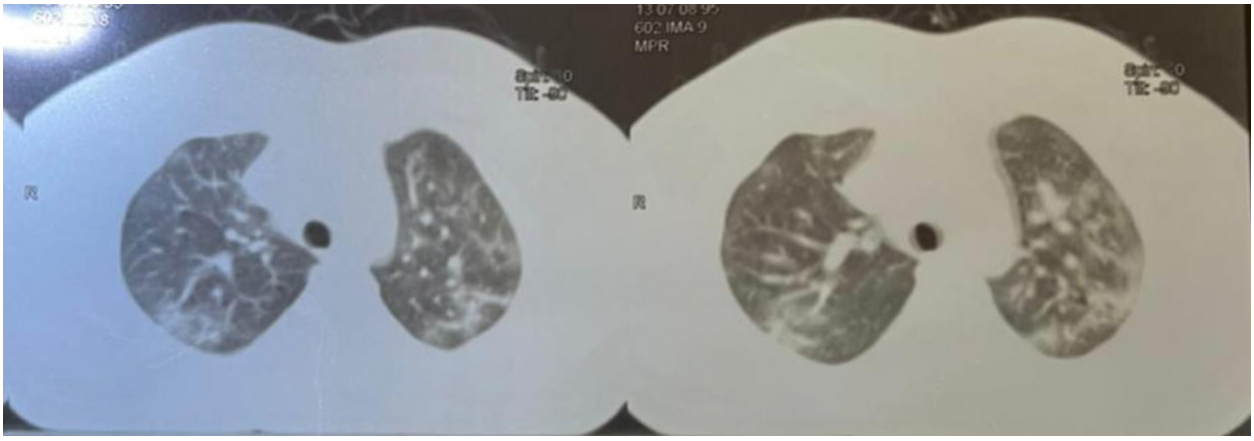

Figure 1: CT of case 2.

Figure 2: CT of Case 3.

patient was switched to oral prednisolone for 3 weeks. Patient saw marketable improvement and was able to go back to normal after 1 month of treatment.

Case 3

45-year-old obese female, with no past medical history, she presented with a 2-week history of irritative cough but no fever, or chills. The patient had developed shortness of breath on exacerbation, and she consulted her family doctor and he advised her to take a covid 19 test which came back positive. The patient remained afebrile, but her shortness of breath continued to increase, to the point where his O_2 stats was 50%. Patient was suspected of post covid 19 syndromes, the patient CT scan showed diffuse fibrosis of both lung fields.

After CT scan showing complete obliteration of lung field, the standing diagnosis was post covid fibrosis. We started her on Prednisolone20 mg PO daily, with 750 mg Levaquin, 10 L of O_2 and watchful waiting, alongside supportive therapy. The patient shortness of breath started to respond positively, after 7 days, her O_2 consumption was dopped to 4 L, that we continued Prednisolone for another 2 weeks, the patient requirement was decreased to 2 L, with mild shortness of breath. Then After 25 days, the patient was off Oxygen while seated, but required it while ambulating, continued on Prednisolone for another week, after 40 days of care patient was largely improved, and post infection X-ray showed clear lung fields a stark difference to her previous CT.

Discussion

We found that case 1 and case 2 they have severe COVID-19 symptoms with extensive fibrosis, and we do not know whether the underlying genetics or general health factors that could have impacted their prognosis, but we do know that their response to high dose steroid, and early antibiotics coverage helped them to recover. Case 1 did express a more severe form of the infection requiring higher O_2 amount, however the early initiation, with IV antibiotics, steroid, and proper supportive measures did quench his symptoms.

The importance of daily supportive care including labs were instrumental because Case 1 developed hyperglycemia, blood glucose had reached 500 mg, which was properly treated with insulin, we further believe that early ambulation whilst still on Oxygen was instrumental in getting case 1 back on his feet, despite the difficulties the infection caused. Both of our obese cases did however have more severe form of the infection, expressed a prolong duration of hypoxemia, required a more intensive steroid intervention. Case 3 presented a very interesting conundrum while she had a relatively mild form of disease symptoms, she had the worst post COVID- 19 infection lung fibrosis, she continues to develop severe hypoxemia, cough, difficulty on ambulation, she was placed on a daily 20 mg dosage of cortisone for 1 month, with supportive treatment, after 3 weeks she showed marked improvement, and the cortisone was tapered off by the end of the month.

Conclusion

Over the course of our 3 cases series, we were able to observe a clinical association between obesity, and COVID-19 symptoms and post infection symptoms. While this is a preliminary outcome on the impact, it does raise questions about disease modifications in patients with obesity, and its impact on response to treatment. We also saw the importance of supportive treatment, in detection of COVID-19 complications, as early detection and intervention made a huge difference in patients' prognosis. Post COVID-19 infection can present a very formidable disease course in some patients, especially with regards to pulmonary fibrosis. Therefore, dormant, or subclinical COVID-19 infections can still cause severe infections and fibrosis, thus recognition and intervention with high dosage steroids alongside IV antibiotics, and supportive treatment may help in recovery of these patients.

References

1. Guan Wj, Ni Zy, Hu Y, et al. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med. 2020; 382: 1708-1720.
2. Salje H, Tran Kiem C, Lefrancq N, et al. Estimating the burden of SARS-CoV-2 in France. Science. 2020; 369: 208-211.
3. Mohammad S, Aziz R, Al Mahri S, et al. Obesity and COVID-19: what makes obese host so vulnerable?. Immun Ageing. 2021; 18: 1.
4. Horby P, Lim WS, Emberson JR, et al. Dexamethasone in hospitalized patients with Covid19. N Engl J Med. 2021; 384: 693-704.
5. Li S, Gong M, Zhao F, et al. Type I interferons: distinct biological activities and current applications for viral infection. Cell Physiol Biochem. 2018; 51: 2377-2396.
6. Watson A, Spalluto CM, McCrae C, et al. Dynamics of IFN-β responses during respiratory viral infection: insights for therapeutic strategies. Am J RespirCrit Care Med. 2020; 201: 83-94.
7. Yuen CK, Lam JY, Wong WM, et al. SARS-CoV-2 nsp13, nsp14, nsp15 and orf6 function as potent interferon antagonists. Emerg Microbes Infect. 2020; 9: 1418-1428.
8. Hadjadj J, Yatim N, Barnabei L, et al. Impaired type I interferon activity and inflammatory responses in severe COVID-19 patients. Science. 2020; 369: 718-724.
9. Agrawal A. Mechanisms and implications of age-associated impaired innate interferon secretion by dendritic cells: a mini-review. Gerontology. 2013; 59: 421-426.
10. Coronaviridae Study Group of the International Committee on Taxonomy of Viruses. The species Severe acute respiratory syndrome-related coronavirus: classifying 2019-nCoV and naming it SARS-CoV-2. Nat Microbiol. 2020; 5: 536-544.
11. Lee S, Kim T, Lee E, et al. Clinical Course and Molecular Viral Shedding Among Asymptomatic and Symptomatic Patients With SARS-CoV-2 Infection in a Community Treatment Center in the Republic of Korea. JAMA Intern Med. 2020; 180: 1447.
12. Siemieniuk RA, Bartoszko JJ, Ge L, et al. Drug treatments for covid-19: living systematic review and network meta-analysis. BMJ 2020; 370: 2980.
13. COVID-19 Treatment Guidelines Panel. Coronavirus Disease 2019 (COVID-19) Treatment Guidelines. National Institutes of Health. https://www.covid19treatmentguidelines.nih.gov/