

## Course of COVID-19 in Uncontrolled Diabetic Smoker Patient: Case Report

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### ABSTRACT

52-year-old diabetic, smoker, man, never hospitalized, exposed to COVID-19 and the course of disease was complicated and not responding to usual COVID-19 treatment.

**Keywords:** Diabetes mellitus; COVID-19

### INTRODUCTION

The studies showed that severity of Corona virus disease (COVID-19) caused by infection with severe acute respiratory syndrome Corona virus 2 (SARs-COV-2) in patients with diabetes mellitus is more prevalent. COVID-19, by itself might predispose individuals to hyperglycemias which might modulate immune and inflammatory response [1].

The following case represented deteriorated complicated course of COVID-19 in diabetic patient.

### CASE DESCRIPTION

52 y-old smoker, obese diabetic man (H<sub>g</sub>A<sub>1c</sub> 9), exposed to COVID-19, a week later, started to experience black tarry diarrhea and mild fever x 1 week, then experienced dry cough, Shortness of Breath (SOB). COVID-19 swab was negative, stayed home, started on hydroxychloroquine and azithromycin. Continued rising fever, worsening SOB, diarrhea and abdominal pain. Two days later, admitted with positive COVID-19 swab, Chest x-ray: bilateral basal infiltrates, received 2L O<sub>2</sub>, hydroxychloroquine and azithromycin. D-dimer was not elevated, continued for 1 week with progressive deterioration of Po<sub>2</sub>, high fever, severe abdominal pain, marked hyperglycemia (400 mmol), Po<sub>2</sub> declined to 90. Put on O<sub>2</sub> 15 L facial mask, worsened SOB, Po<sub>2</sub> (77-80). Transferred to tertiary center, started high flow O<sub>2</sub>, solu-Medrol, anticoagulants, insulin. Became extremely deconditioned, with 20 lb loss, with only bed toileting, continued steroids for two weeks. His blood picture showed pancytopenia with marked thrombocytopenia (40000 count).

hematology consult recommended follow up. Platelet continued to drop. Restarted on prednisone 60 mg qd, platelet started to rise very slowly. Started bed side physical therapy, continued hospitalization for 1 month, discharged home on oxygen 2 L nasal canula ad therapy with continuation of oral prednisone 40 mg with slowly tapering dose over another month and anticoagulants. His platelet reached 80000 after two weeks from discharge. Returned to his baseline in another month.

### DISCUSSION

COVID-19 with obese uncontrolled DM, smoker x (20 years) man. Never diagnosed as COPD. Prolonged disease course complicated with severe thrombocytopenia.

Hussain et al. reported that diabetes lead to significant predictor of morbidity and mortality, chronic inflammation, increased coagulation activity, immune response impairment and potential direct pancreatic damage by SARS-COV2 [2].

Kumar A et al. reported that diabetic patients have two folds in mortality and severity of COVID-19 as compared to non-diabetics [3].

Muniyappa et al. suggested the potential mechanisms that might increase the susceptibility of diabetic patients to worse COVID-19 infection including; higher affinity cellular binding and efficient virus entry, decreased viral clearance, Diminished T-cell function, increased susceptibility to hyperinflammatory and cytokine storm syndrome, in addition to presence of associated cardiovascular disease [4].

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Interaction between COVID-19 and diabetes mellitus set up a vicious cycle of cytokine storm and cascade. COVID-19 leads to worsening of dysglycemia and thus exacerbation of the severity of COVID-19 [5].

Tadic et al. suggested in their study that DM leads to higher cellular binding, decreased viral clearance, diminished T-cell function, more cytokine storm with cascade of Interferon gamma, IL17, IL21, IL22, IL6, elevated C-reactive protein, sedimentation rate, D Dimer and fibrinogen [6].

All these documented studies can explain why the course of this case is an exaggerated, complicated course of COVID-19 although, the beginning was not that severe. In addition to the presence of smoking.

As regards effect of smoking and course of COVID-19, Van Zylsmit et al. reported that smoking increase susceptibility to infection, might be to upregulation of the angiotensin converting enzyme 2 ACE2 receptor, the main receptor used by severe acute syndrome corona virus 2 (SAR-COV-2) to gain entry to host mucosa and cause active infection. Smoker have increased gene expression of ACE2 than previous smoker or non-smoker [7].

## CONCLUSION

Uncontrolled DM complicates the course and prolongs recovery of COVID-19, and the presence of smoking even without

history of overt COPD, might play a role in complicating the course and delaying the recovery from COVID-19 infection.

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