Case report

Coronavirus Disease 19 (COVID-19) in a Patient with Chronic HCV while on Direct-Acting Antiviral Therapy with a Worse Prognosis

Abstract:

Introduction:
The world is suffering a major global health pandemic caused by a new strain of the coronavirus (COVID-19). Herein, we encountered one case with COVID-19 and chronic HCV while on Direct Acting Antiviral Therapy.

Case presentation:
A 58-year male patient had chronic hepatitis C (HCV) without liver cirrhosis. He was on Direct- Acting Antiviral Therapy for HCV (DAAs) in the form of Sofosbuvir 400 mg daily and Daclatasvir 60 mg daily (on his third month). The patient developed acute respiratory symptoms suggestive of pneumonia. Oropharyngeal swab for COVID-19 was positive as detected by real-time polymerase-chain-reaction (PCR) assay. The treatment for COVID-19 was given according to the Ministry of Health Protocol in addition to oxygen therapy with the continuation of his anti HCV therapy. His symptoms and oxygen saturation progressively deteriorated. The patient died despite supportive measures.

Conclusion:
Clinicians should suspect a worse prognosis of COVID-19 in chronic HCV patient despite supportive therapy for COVID -19. The efficacy of Anti HCV therapy as protective or therapy against COVID-19 needs clinical trials.

Keywords: COVID-19; SARS-CoV-2; HCV; Coinfection; DAAs, Case report.

1.INTRODUCTION:

The World Health Organization (WHO) was informed about the emergence of a new virus from CORONA viruses' family on December 31st, 2019 [1]. This virus emerged from Wuhan City of Hubei Province of China and named by WHO as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) or (COVID-19) [2, 3, 4]. WHO declared that COVID-19 is considered a pandemic on 11 March 2020 [5]. Globally, 7, 941, 791 cases of COVID-19 have been reported with 434, 796 confirmed deaths as stated in WHO report on 16 June 2020 [6]. Risk factors for poor prognosis are older age, male sex and presence of comorbidities (obesity, diabetes, heart disease, lung disease, kidney disease) [7].

It is unclear to what degree chronic liver diseases could be considered as risk factors, due to lack of appropriate studies [8]. However, patients with chronic liver disease, especially viral hepatitis B and/or C, may be more vulnerable to liver damage from COVID-19, as was reported with SARS-CoV, but supporting data are limited [9].

To the best of our knowledge, the coinfection of COVID-19 and chronic HCV cases on treatment may occur in Countries with high prevalence of HCV, but not reported. Here, we present one case with occurrence of COVID-19 infection in chronic HCV case while on Anti-HCV. Treatment.

2.PRESENTATION OF CASE:

A 58-year- old male patient had chronic hepatitis C without liver cirrhosis. He was on hepatitis C treatment in the form of Sofosbuvir 400mg daily and Daclatasvir 60 mg daily (on his third month). The patient developed acute respiratory symptoms in the form of high-grade fever and cough four days prior to presentation followed by shortness of breath 1 day prior to her admission to the Emergency Department on 15th of May 2020.

On physical examination at the time of admission, the patient had fever 38.5^0C, had an arterial blood pressure of 150/80 mmHg, a heart rate of 100 beats/min, respiratory rate of 40 breaths/min and oxygen saturation of 90% on room air. Chest examination revealed bilateral crepitation. On admission, urgent complete blood gas, and routine blood tests, were done. The arterial blood gas on room air showed a PaO2 of 51 mmHg, PaCO2 of 29 mmHg, HCO3 of 19 mmol, pH of 7.45. Acute respiratory failure type 1 was suggested. Oxygen therapy was given using venturi mask 50%.

Urgent blood tests revealed the following: hemoglobin level of 12.9 g/dL, leucocyte count of 13.5 (x 10^3/ul), with 75 % neutrophils, 20.0% lymphocytes, and 2.0% monocytes, platelet count of 279 (x10^3/ul.) Random blood sugar 150 mg /dL. Na 134 mmol/L (136-145). calcium 8.4 mg/dl (N 8.6-10.2), K 3.7 mmol/L (N 3.5-5.1). creatinine 61 µmol/L (N 66-106), Troponin 0.03 (N up to 0.05), CPK-MB 68 U/L (N < 25), and D dimer (0.8) mg/L (N up to 0.55). ECG sinus tachycardia. Abdominal Ultrasonography showed mild hepatosplenomegaly and normal both kidneys. High resolution CT chest revealed multiple ground glass alveolar opacities (more evident on bilateral lower lung lobes) goes with diagnosis of COVID -19 **(Figure 1)**. Oropharyngeal throat swab sample for COVID-19 was positive as detected by real-time reverse-transcriptase– polymerase-chain-reaction (RT-PCR) assay.

The patient was admitted to Al-Rajhi Liver Hospital (COVID-19 quarantine hospital) on the 16th of May 2020 after confirmed positive PCR for COVID -19. Treatment was given according to Ministry of Health Protocol (Hydroxy chloroquine 400 mg twice per day for 1 day then 200 mg every 12 hours, Vitamin C 1 gm per day, Zinc 50 mg per day, Antipyretics as needed) in addition to oxygen therapy on Venturi mask 50% and maintained on his anti HCV therapy.

Complete laboratory investigations after admission was as follow, ferritin 1125 ng/ml (N 22-322), Fibrinogen level 3.34 g/L (N 2-4), High sensitive C-reactive protein (CRP) 202 mg/L (N 0-6). Triglycerides 74 mg/dl(50-150), PT 13.7 Second, Conc 72%, INR 1.14, Total bilirubin 8.5 umol/l (N 5-21), total protein 61 g/L (N 64-83), Albumin 21g/L(N 34-50), AST 45 U/L(N <34), ALT 62 U/L (N 10-49), GGT 54 U/L (N <63),ALP 60 U/L (N 46-116).

Two days after hospital admission, follow up Hb 13.4 g/dl, PLT 279(x10^3/ul), WBCs 18.4 (x10^3/ul), lymphocytes 17% (N 20-45), ESR 1st hour 80 mm (N 3-5), 2nd hour 120 mm (N 7-12). Creatinine 61 µmol/l (N 44-106), Follow up LFT, Total bilirubin 13.6 umol/l (N 5-21), total protein 59.8 g/L (N 64-83), Albumin 27.7 g/L (N 34-50), AST 43.6 U/L (N <34), ALT 39.6 U/L (N 10-49), GGT 40.1 U/L (N <63), ALP 72.4 U/L (N 46-116). Ferritin 2525 ng/ml (N 22-322). D dimer 1.83 mg/L (N up to 0.55). Linezolid 600 mg IV every 12 hours and prophylactic dose of low molecular weight heparin were added.

One day later, worsening of oxygen saturation, so the patient was shifted to noninvasive mechanical ventilation. There was no improvement on noninvasive mechanical ventilation and the patient died on the fourth day of hospital admission.

3.DISCUSSION:

We have reported this case with specific interest due to coinfection of SARS-CoV-2 and Chronic HCV while on treatment which is not common. The essential factor that discriminates who complains of minor symptoms of COVID 19 and who will be very sick is their baseline state of health. Hepatitis C is a serious, pre-existing health condition. Therefore, a person who is infected with Hepatitis C is more likely to suffer serious illness and confront a difficult, if not a fatal experience from a COVID 19 infection than a person who does not have Hepatitis C (10).

Several studies stated that Sofosbuvir and other Direct Acting Antivirals (DAAs) could inhibit COVID 19 replication (10). Coronaviruses are positive-strand RNA viruses with conserved polymerase, so SARS-CoV-2 RNA-dependent RNA polymerase (RdRp) is suppressed by Sofosbuvir. So, it is hypothesized that SARS-CoV-2 infection could be susceptible to Sofosbuvir (11). Sofosbuvir, Ribavirin, and Remdisivir can be used against COVID 19 with promising results (12).

However, in our case despite he was on Sofosbuvir and Daclatasvir therapy, he gets infected with COVID-19 and his prognosis was poor with death at the end despite supportive therapy for COVID -19 and DAAs.

4.CONCLUSION: Clinicians should suspect a worse prognosis of COVID-19 in chronic HCV patient despite supportive therapy for COVID-19. The efficacy of Anti HCV therapy as protective or therapy against COVID-19 needs clinical trials.

5.REFERENCES:

1. Zhu, N., Zhang, D., Wang, W., Li, X., Yang, B., Song, J., et al., 2020. A novel coronavirus from patients with pneumonia in China, 2019. New. Engl. J. Med. 2020 January 24th (Article in Press).
2. Wu F, Zhao S, Yu B, et al. A new coronavirus associated with human respiratory disease in China. Nature. 2020;579(7798):265–269. DOI:10.1038/s41586-020-2008-3.
3. Wang Chen, Horby Peter W, Hayden FG, Gao GF. A novel coronavirus outbreak of global health concern. The Lancet. 2020; 395(10223):470–473.
4. Coronavirus Outbreak. Available at: https://www.worldometers.info/coronavirus/. (Accessed 5 April 2020).
5. World Health Organization. Director-General's opening remarks at the media briefing on COVID19—11 March 2020". https://www.who.int/dg/speeches/detail/who-director-general-s-openingremarks- at-the-media-briefing-on-covid-19---11-march-2020. Accessed April 13, 2020.

6. World Health Organization. “Coronavirus disease (COVID-2019) situation reports. Situation report - 148 . Available at: https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports. Accessed June 16, 2020
7. AASLD. Clinical Insights for Hepatology & Liver Transplant providers during the COVID-19 Pandemic. https://www.aasld.org/sites/default/files/2020- 04/AASLD-COVID19 Clinical Insights-4.07.2020-Final.pdf. Accessed April 13, 2020.
8. Boettler T, Newsome PN, Mondelli MU, Maticic M, Cordero E, Cornberg M,Berg T, Care of patients with liver disease during the COVID-19 pandemic: EASL-ESCMID position paper, JHEP Reports (2020), doi: https://doi.org/10.1016/j.jhepr.2020.100113.
9. Xu L, Liu J, Lu M, Yang D, Zheng X. Liver injury during highly pathogenic human coronavirus infections. Liver Int 2020 Mar 14. https://doi.org/10.1111/liv.14435
10. Jefferys G. Hepatitis C and the COVID 19 Corona Virus. March 20, 2020. https://www.genericchepatitiscdrugs.com/hepatitis-c-and-the-covid-19-corona-virus/.
11. Sayad B, Sobhani M, Khodarahmi R. Sofosbuvir as Repurposed Antiviral Drug Against COVID-19: Why Were We Convinced to Evaluate the Drug in a Registered/Approved Clinical Trial? Archives of Medical Research. Accepted April 23, 2020(ARCMED_2020_488).
12. Elfiky AA. Anti-HCV, nucleotide inhibitors, repurposing against COVID-19. Life Sciences; 248, 1 May 2020. https://doi.org/10.1016/j.lfs.2020.117477.

Figure 1 : High resolution CT chest findings suggestive of COVID-19.