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Research Article

COVID-19 IN A PATIENT WITH HEPATITIS- A: A DIAGNOSTIC & THERAPEUTIC DILEMMA

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ABSTRACT

Rationale: COVID-19 also known as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV 2) is a virus known to cause pneumonia like clinical picture along with various respiratory and cardiovascular symptoms that exhibits thrombogenic activity and inflammatory events.
Disease prognosis worsens with co-morbidities & other pre-existing conditions. currently, the treatment includes providing supplemental oxygen therapy, supportive and symptomatic care along with experimental Anti-Viral drugs and Corticosteroid therapy. Ultimately, mechanical ventilation in severe cases admitted to the intensive care unit.
Patient complaints: A 44 year old male with H/O hyperlipidemia who presented with symptoms of worsening fever, mild icterus, breathlessness, diarrhoea and abdominal pain
Diagnosis: Hepatitis A was confirmed positive on serological testing along with CXR showing patchy ground glass opacities in both lung bases and HRCT Chest showing multiple ill-defined sub plural opacities with ground glass attenuation showing crazy paving pattern. These findings were consistent with COVID-19. However, RT-PCR revealed a negative result prior to HRCT Chest.
Interventions: Patient was unwilling to undergo hospital admission & second RT-PCR. Thus, after high risk consent from patient and his relatives, he was started on supplemental oxygen therapy, Azithromycin, Favipiravir, Dexamethasone, LMWH, along with other supportive treatment including zinc, glutathione. Vitamin C, Vitamin D3 and a Multivitamin. The patient completed the full course of treatment advised in home isolation without deterioration.
Outcome: The patient's health improved with near complete resolution of COVID-19 changes on HRCT, remission of all symptoms and return of all haematological parameters to baseline.
Lessons: The simultaneous presence of two viral infection like Hepatitis-A & COVID-19 can lead to rapid deterioration of patient's condition and progression of their symptoms. Also, the presence of two viral infections can lead to further complexity due to overlapping symptoms of the two infections causing a diagnostic challenge and thus delaying diagnosis.
Anti-Viral Therapy (Favipiravir) approved for moderately symptomatic COVID patients are known to elevate liver enzymes, which is also seen as a result of Hepatitis-A thus making therapeutic decisions sceptic. Therefore, regular monitoring of Liver enzymes is key to monitor disease prognosis and prevent further complications which is not routinely done in standalone COVID-19 cases.
Early diagnosis and intervention on display of COVID-19 symptoms can prevent deterioration and need for mechanical ventilation and also prevent extensive lung fibrosis post COVID-19.
RT-PCR shows significant false negative results and should not be considered as the only diagnostic modality for COVID-19. Diagnosis should comprise of RT-PCR along with HRCT chest and must be co-related clinically to establish a strong diagnosis of COVID-19 with the use of Chest X-Ray as an inexpensive radiological Screening test.

INTRODUCTION

COVID-19 (SARS-CoV2) belonging to the Beta Coronaviridae family is novel infectious disease discovered towards the end of 2019 allegedly in a wet market in Wuhan, China. Earlier in 2020, this virus was declared to spread via human to human transmission, primarily through droplets which lead to a mass scale spread and a pandemic being declared soon after in March 2020. It is genetically similar to the SARS Coronavirus which was responsible for the SARS outbreak in 2002.

Individuals with COVID-19 can present with a influenza like illness and respiratory tract infection demonstrating fever (78%), cough (57%), fatigue (31%), and/or Shortness of breath especially in hospitalised patients where 19% required non-invasive ventilation and 9% required invasive ventilation and 2% required extra corporal membrane oxygenation[1]. Other

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common symptoms like diarrhoea, myalgia, anosmia, ageusia, etc are also seen.

Patients with COVID-19 have also exhibited thrombogenic and inflammatory changes and events thus also making it an acute- immunogenic- thrombogenic- inflammatory viral disease pandemic.[2] As of the date of this case study, there is no steady antiviral or immunomodulatory treatment for SARS-COV2 (COVID-19).

Hepatitis-A virus is the most common cause of acute hepatitis worldwide. It is primarily transmitted through faecal oral Route via contaminated food or water or by contact with an infected person. As per WHO it is estimated that in the year 2016, Hepatitis-A virus has resulted in 7134 deaths[3]and around 1.3 million cases of HAV infection are seen yearly.[4] HAV is know to rarely be fatal, however it can cause debilitating symptoms and fulminant hepatitis (Acute liver failure) which can prove be fatal. Classic symptoms of HAV are fever, diarrhoea, icterus, dark coloured urine, loss of appetite, Anorexia[5] etc. There is no specific treatment for HAV as most cases are selfresolving with supportive treatment. However a vaccine exists today, available internationally which is protective against the virus.

While HAV by itself may resolve in majority of the population, when occurs with another virus like COVID-19 may lead to complications both by the direct effect of the letters hepatotoxic drugs as well as the indirect effects of a patient's immunogenic status.

Case presentation

A 44 year old, Indian Male with a medical h/o hyperlipidemia who is non-alcoholic, non- smoker, with a positive family history of COPD and hypercholesterolemia contacted us, his primary healthcare physicians with a 3 day history of worsening high grade fever (102°F) with chills partially relieved with PCM 500mg SOS, mild icterus, diarrhoea, myalgia. He had a h/o travel to a red zone area infected with high volume of COVID-19 patients as well as h/o consumption of stagnant water from an unknown source. (day3) Based on the symptoms, he was asked to do the following tests:

CBC, LFT, Dengue NS1, Malarial parasite on Peripheral blood smear, Dengue IgG, WIDAL for Typhoid, MAT for leptospirosis and Serology Test for Hepatitis-A and E.

His vitals were as follows: Temp 101.4° F, Pulse= 92 Bts/min, Blood Pressure =110/72mmHg &SpO2= 96% on room air.

He was prescribed symptomatic treatment on day 3 which gave him mild relief. His test results were as follows: his CBC showed leukopenia as well as thrombocytopenia along with elevated SGPT & SGOT levels. His Serology for HAV was positive while all other tests done were negative. He was started on supportive care with being prescribed PCM 500 TDS, Multivitamins, ORS, Probiotics, Liv52 (Herbal liver protective medication) His condition remained stable for the next 2 days after which he developed a worsening fever, mild dry cough and Shortness of breath on day 6. Fresh CBC and LFT's along with Chest-Xray and RT- PCR was ordered on day 7 and it was reported on day 8 where CXR showed patchy ground glass opacities in both lower zones whereas RT-PCR (nasopharyngeal swab) was negative for SARS-CoV2. Patients PCM dose was increased to 650mg TDS on Day 8 and HRCT chest was ordered on Day 9 along with D-Dimer, CRP, Ferritin, LDH. Patient developed breathlessness on Day 9 with SpO2 drop to 89% on room air. HRCT showed multiple ill-defined sub pleural opacities with ground glass attenuation in Bilateral lung parenchyma with lower lobe predominance and sub segmental atelectasis seen in lower lobes. These findings were classical for COVID-19 pneumonia. After seeing these CT findings a new RT-PCR was advised, however patient declined a repeat swab test. The inflammatory markers done along with the CT were significantly raised with D-Dimer at 1050 ng/ml FEU, CRP at 38.2mg/L and ferritin at 837.49 ng/ml. Patient also developed breathlessness the same day with a drop in saturation to 89% on room air. He was started immediately on Supplemental oxygen therapy at 5L/min with a simple oxygen face mask resulting in a spontaneous improvement in saturation to 98% on 5L/min O2.

He was also asked to monitor & chart his oxygen saturation on room air and on O_2 every 2 hourly.

The Patient wished to be treated at home and declined to get admitted to a hospital, therefore a high risk consent was taken and patient was started on the following medications (day 9) :-

- Tab Dexamethasone 6mg OD
- Tab Favipiravir 1800mg BD on Day 1 of medication f/b 800mg BD for the next 7 days.
- Inj. Enoxaparin SQ 0.4ML OD for 7 days
- Tab L- Glutathione 500mg OD for 15 days
- Tab Zinc Sulphate 200mg OD for 15 days
- Tab Vitamin C 500mg OD for 15 days
- Tab Multivitamin B complex OD for 15 days

Fresh CBC and LFT's were done on day 11 which showed persistent leukopenia and thrombocytopenia and SGOT at 144 IU/L and SGPT 269 IU/L. However patients symptoms improved with fever now being under control and saturation being maintained at 96% on 4L/min O2 and 92% on room air and other symptoms such as diarrhoea and myalgia under control.

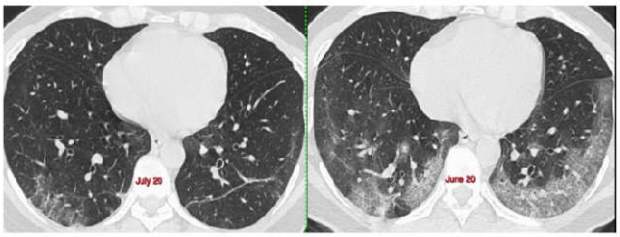
Serial CBC and LFT were done every 5 days to monitor patients liver enzymes and haematological parameters throughout medical therapy as well as to monitor disease prognosis.

On Day 17 repeat D-DIMER reached its peak at 4625 ng/ml FEU. However, CRP & Ferritin were normalised to 1.2 mg/L and 381.1 ng/ml respectively.

Patient's symptoms had resolved by Day 17 after 7 days of medication and eventual weaning off the supplemental Oxygen and he was maintaining saturation of 98% on room air.

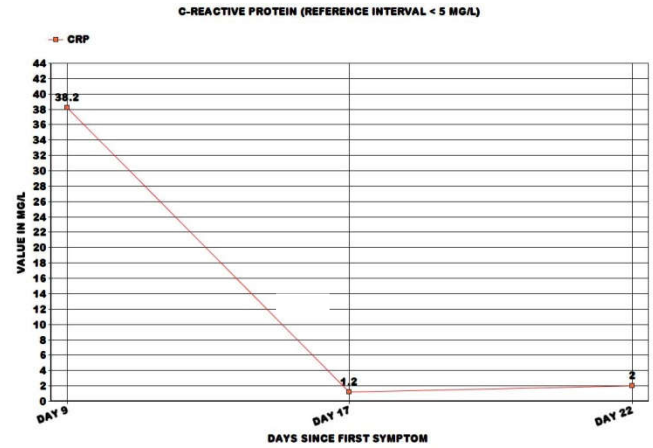
Patient was advised to continue monitoring his symptoms and oxygen saturation on Room air 4 hourly and report S.O.S. if saturation dropped below 94%. Repeat HRCT chest done on Day 17 which showed significant improvement with residual ILD changes as compared to the first HRCT chest.

Further serial monitoring of CBC, LFT and inflammatory markers indicated their return to baseline over the next 7 days. Follow up testing 3 months post COVID-19 and HAV status reported a normal CBC, LFT and Inflammatory marker values.

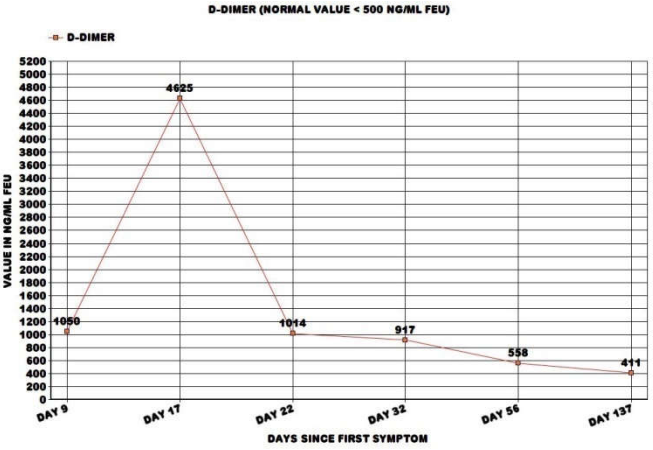


DAY 17DAY 9

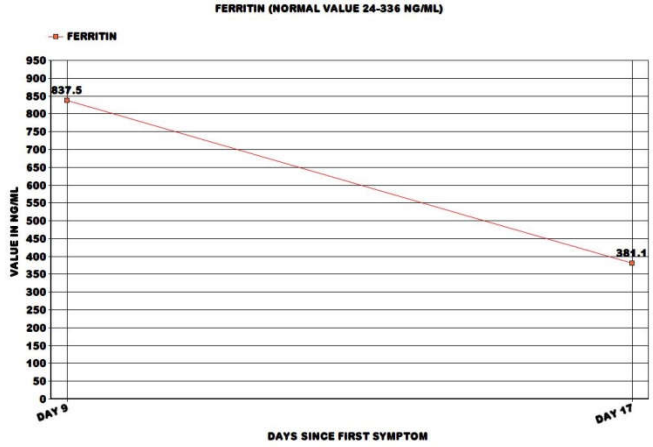
Figure 1 HRCT Chest Before and After



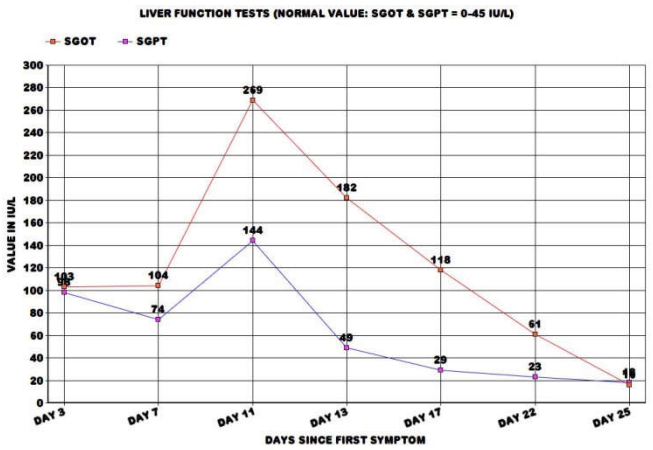
Graph 1 Change in CRP levels



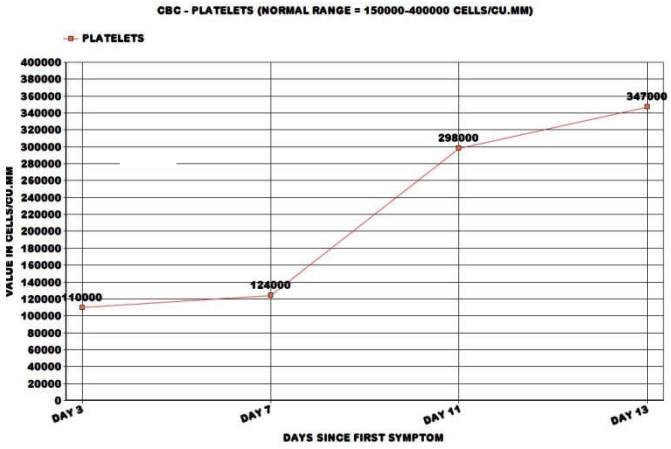
Graph 2 Change in D-Dimer levels



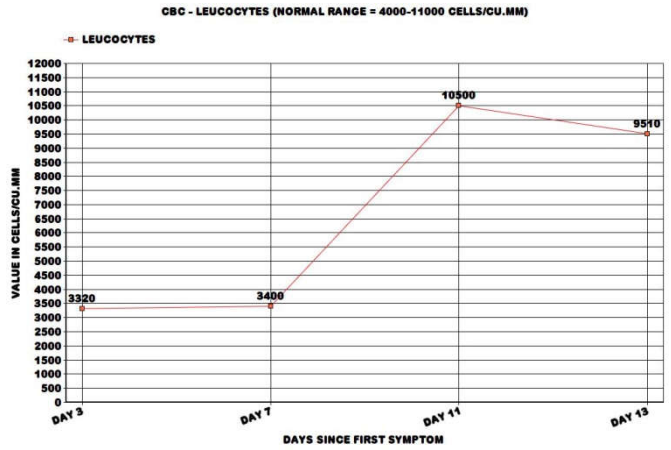
Graph 3 Change in Ferritin levels



Graph 4 Change in LFT values



Graph 5 Change in Platelet values



Graph 6 Change in Leucocyte values

METHODS

For analysis, we collected the patient's medical records, which included clinical characteristics, laboratory parameters, chest CT imaging, treatment approach, and clinical outcome. This independent case study was done at General Practitioner level. Treatment was given at patient's residence and study was conducted via home visits. Informed consent was pre-obtained.

DISCUSSION

COVID-19 is a new disease, caused by a novel coronavirus SARS-CoV2. Reported confirmed cases have ranged from mild symptoms to severe illness and death. COVID-19 patients can present with a influenza like illness and respiratory tract infection demonstrating fever (78%), cough (57%), fatigue

(31%), and/or Shortness of breath especially in hospitalised patients.

Fever is the most common symptoms of COVID-19 and is the first symptoms that developed in our patient along with mild icterus these symptoms made us suspect an infective ethology most likely to be hepatic in nature which in turn made us workup the patients LFT, Hepatitis serology markers along with his haematological fever profile. This initially led us to diagnosing the patient with viral hepatitis which was confirmed with a positive serology for Hepatits-A and history off consumption of stagnant water from unknown source. As per HAV treatment protocols patient was then started off on supportive therapy to control all symptoms along with serial monitoring of liver function tests to check for disease prognosis. Failure to control symptoms along with development of consequent shortness of breath and a positive history of trade to endemic area infected withCOVID-19 patients made us suspect an additional provisional diagnosis of a respiratory infection most likely COVID-19 due to the given pandemic.

This diagnosis was supported by a CXR showing mild patchy ground glass opacities in the lower zones bilaterally[6] most likely due to an infective etiology along with elevated CRP & Ferritin[7]. This provisional diagnosis was contradicted by a negative SARS-COV2 RT-PCR however, confirmed on HRCT which showed typical COVID findings such asground glass opacities and crazy paving pattern[8]. This gave us an impression that the RT-PCR test report was probably a false negative which was highly possible since False negative rates are highest within the first 5 days after exposure (up to 67%), and lowest on day 8 after exposure (21%)[9], however this could not be confirmed due to lack of patients consent for a second RT-PCR testing.

High risk consent was taken as patient also denied hospitalisation as recommended observing a drop in saturation and deterioration of status. He was then started off on Dexamethasone which was known to lower 28 day mortality than this who didn't receive it in then placebo group[10] And Favipiravir an antiviral that inhibits RNA dependent RNA polymerase which ultimately prevents viral transcription and replication therefore in COVID-19 facilitating rapid viral clearance and potent in vitro activity[11]. This was done with close regular monitoring of his LFTs in view of his HAV status along with administration of antivirals having a hepatotoxic side effect. The patient was also started on InjEnoxaparin due to an elevating D-Dimer, possibly caused by the thrombogenic effect of COVID-19 caused due to Endothelial dysfunction, activation of the renin-angiotensin-aldosterone system (RAAS) with the release of procoagulant plasminogen activator inhibitor (PAI-1), and hyperimmune response with activated platelets seem to be significant contributors to thrombogenesis.[12]Low Molecular Weight Heparin not only plays its role as an anticoagulant in the high thrombogenic status of COVID-19 but also benefits patients' with its other non-anticoagulant effects such as Inhibition of Heparanase activity, responsible for endothelial leakage, Neutralisation of chemokines and cytokines; Interference with leukocyte trafficking and Reducing viral cellular entry[13].

Home supplemental oxygen therapy via Oxygen Concentrator and simple O_2 face mask to counter the fall in saturation on room air was given at appropriate quantity based on hourly blood oxygen saturation monitoring. Oxygen therapy in COVID-19 not only brings up a mild drop in blood O_2 saturation levels to normal but also helps in disrupting viral replication, improving antiviral immune response and reduce upregulation of ACE2 expression[14].

The Patient was also started on L-Glutathione which in limited studies was show to be beneficial both in COVID and Hepatitis. In COVID-19, Glutathione was seen to arrest symptomatic progression by preventing activation of NF-κB (nuclear factor-kappaB) which is known to be a pro-inflammatory mediator associated with development of ARDS cytokine storm syndrome in patients with COVID-19 pneumonia. A study involving the use of Glutathione showed that it reduces the serum levels of asparaginic acid transaminase, alanine aminotransferase, total bilirubin, total bile acids, haluronic acid, collagen IV, laminin, transforming growth factor-β_1, tumour necrosis factor-α, interleukin-6, and interleukin-8.[15-17]and may also protect it from oxidative stress and ischemic injury[18].

A complete Multivitamin was prescribedas Adjunct medical therapy along with Vitamin C and Zinc supplementation, both of which are known to support the immune system, shorten the disease duration and improve outcomes in respiratory infections like pneumonia[19-21].Regular CBC's which initially showed Leukopenia and thrombocytopenia, now had gone back to their biological reference range along with transaminase levels reducing back towards their baseline. The patient reported of marked symptomatic improvement along with maintaining oxygen saturation in room air as the oxygen was gradually weaned off. The D-Dimer kept increasing despite patient taking subcutaneous Enoxaparin and however later stabilised[22] with gradual decrease over period of time as HRCT scan showed marked improvement compared to previous imaging. Other inflammatory markers such as CRP and Ferritin returned back to baseline.

The clinical manifestations of Hepatitis-A led to the masking of symptoms of COVID-19 causing the delay in its diagnosis. This delay produced a diagnostic challenge leading to a prolonged disease course without COVID specific treatment ended up causing symptoms such as shortness of breath which is clinical picture of COVID-19 and not Hepatitis-A. The overlapping of these two viral diseases led to a delay in diagnosis of COVID19 causing a diagnostic challenge and a delay in COVID specific therapy. The presence of hepatitis a not only led to the delay diagnosis of COVID-19 but also proved to be a therapeutic challenge as well, in the form of elevated LFT's hampering the un restricted use of Favipiravir and other Anti-Viral drugs due to their hepatotoxic side effects. This called for the need of regular LFT's which not only raised the cost of treatment but also the duration of treatment.

CONCLUSION

Overlapping diseases seen along with COVID-19 (Eg: Hepatitis-A) always pose a significant diagnostic dilemma especially when there is a similar symptomatic profile which may cause a delay in the accurate diagnosis of COVID-19 & receiving treatment for it which in turn may lead to progress of symptoms to that of shortness of breath and others belonging to that of the moderate to severe symptom category. In such cases especially in a COVID-19 global pandemic, a Chest X-ray must be done as a low cost screening modality in all patients showing symptoms of not only COVID but any other

disease "(except: in pregnancy or if contraindicated otherwise). COVID-19 may also by itself present as Acute Hepatitishence timely testing to rule out COVID is quintessential. Also, monitoring of regular monitoring of LFT's after being diagnosed with COVID-19 helps rule out COVID-induced hepatitis[23].

RT-PCR testing for COVID-19 through nasopharyngeal swab holds a significantly high risk of being false negative, therefore when patients exhibit symptoms of COVID-19 such as fever, cough& breathlessness, a positive HRCT with classical COVID-19 findings should be considered diagnostic despite a first negative RT-PCR swab. In such cases where COVID symptomology is classic, an HRCT Chest is a more sensitive diagnostic tool than RT-PCR Swab testing. Treatment of COVID-19 should not be delayed by waiting for a second RT-PCR especially in presence of another infection due to chances of worsening prognosis and symptoms caused by the delay in RT-PCR diagnostic reporting over HRCT Chest which is reported immediately.

Favipiravir and Dexamethasone therapy is shown to help in patients belonging to moderately symptomatic COVID positive category with reduction of symptoms and marked improvement in inflammatory markers and HRCT Chest films over a period of time. Other novel treatment approaches like the use of Glutathione should be given a consideration as additional low cost therapy to standard available COVID therapy which may facilitate viral clearance especially in cases of contemporaneous infections with two or more Viral diseases. Early home supplemental oxygen therapy is key to arrest respiratory status deterioration and may eventual void the need of invasive ventilation if used appropriately with other standard treatment used for COVID19.

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