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Case Report

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Acute viral hepatitis caused by COVID-19

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Introduction

Since December 2019, the coronavirus is rolling in the news. A novel virus, later Classified as SARS-COV-2 was identified as a causative agent of life threatening pneumonia. Disease outbreak started in Wuhan, China and infected people of all age groups in every continent on this planet Earth [1].It was initially called a public health emergency of international concern. On March 11th 2020, World Health Organization (WHO) reclassified the situation as pandemic.

The spectrum of typical respiratory illness varies from asymptomatic, mild to moderate like cough, fever, body aches to life threatening pneumonia [2]. The damage caused by the SARS-COV-2 is not limited to the lungs only. There are several large scale studies in progress to assess the pathogenicity of the virus on other organ systems in the body like liver, kidneys, brain and hematological system [3]. Following is the case report of a lady who had a significant new liver damage after being diagnosed with COVID-19 virus. We will also be discussing the mechanism of liver damage by using the data and available case reports from The Fifth Medical Center of PLS General Hospital, Beijing, China.

Case presentation

A 70 years old lady was admitted to the emergency department with main complaints of sudden onset of confusion and dysphasia. On admission, there was no history of fever, sore throat, cough, shortness of breath, headache, abdominal pain, nausea or vomiting, loose stools, weight loss or any urinary symptoms. She was living with her husband and there was no history of drug overdose, any recent illness or sick contacts. She was given a surgical mask as per the hospital protocol on entering the Red Zone of the department. Her Past medical history includes Aortic Regurgitation, Atrial Fibrillation, Pulmonary Fibrosis, HTN, LBBB, Osteoarthritis, Lumbar Spondylosis, Multiple Sclerosis and Depression. There were no known allergies and her regular medications include pregabalin, bisoprolol, apixaban, mirtazapine, lansoprazole, laxido and co-codamol when needed. Her recent outpatient blood tests were 4 months old and they were all normal. On admission, her temperature was 36.6, respiratory rate of 14, pulse of 87 and blood pressure of 140/90. She had warm peripheries and mild pedal edema with no rashes or lymphadenopathy. She was slightly jaundiced and her abdomen was soft and nontender with a palpable bladder. There was a faint diastolic murmur in the aortic area and both lungs were clear.

Blood test results were as follows: WCC 9.6 10^9/L, Hb 132 g/L, Platelet Counts 237 10^9/L, Total Bilirubin 8 µmol/L (<21), AST 20 IU/L (< 32), ALT 21 IU/L (<33), Alkaline Phosphatase 102 IU/L (30-130), Serum Albumin 36 g/L (35-50), PT 15.1 Sec (N 12), CRP 21, Urea 7.3 mmol/L (2.5-7.8), Creatinine 76 µmol/L (46-92), Sodium 121 mmol/L (133-146), Potassium 5.1 mmol/L (3.5-5.3), eGFR >90 mL/min. Chest X ray on admission showed right sided consolidation with some chronic lung changes. CT head revealed rounded lesion in the left occipital lobe with the differential diagnosis of infarct or a space occupying lesion. She was diagnosed with stroke and aspiration pneumonia for which she was started on the appropriate treatment. On day 2 of the hospital admission, she spiked a temperature of 38.8°C with SpO_2 95% on 2 L of oxygen through nasal specs. A repeated Chest Xray showed that consolidatory changes had extensively increased. She was tested negative for SARS-COV-2 twice and atypical pneumonia screen as well. There was no growth on the preliminary reports of blood and urine cultures. Her repeated liver function tests were deteriorating significantly as mentioned below:

Bilirubin	39 µmol/L	<21
ALT	1325 U/L	<33
ALP	149 U/L	30-130
Protein	54 g//L	60-80
Albumin	30g/L	35-50

Viral markers for Hepatitis A, B, C and E along with autoimmune liver screen came back negative. Also, Serum Caeruloplasmin and a-1-anti-trypsin were within the normal limits. Other bloods results from the Liver Screen are: LDH 1033, TSH 1.04, T_4 21.4 (N), HbA1c 26 (N<45).

There were no hepatotoxic drugs on the prescription chart and no history of any herbal medication. Her confusion and slurred speech completely resolved but there was jaundice with multiple bruises and generalized edema all over the body with severe fluid retention in the subcutaneous tissue secondary to hypoalbuminemia. Synthetic functions of the liver were worsening as evident by Prothrombin time of 25.1 seconds and serum albumin of 26 g/L.

Her oxygen requirement increased remarkably over the next 24 hours and she was not well enough for a liver Ultrasound. Another repeated CXR showed bilateral massive infiltration. We noted that a CT Scan of her abdomen was done in 2016 which showed normal solid organs. Subsequently, a third throat swab of COVID-19 came back positive. Keeping the complex medical background in mind, it was mutually decided that resuscitation and invasive ventilation is not in the best interest of the patient. She was transferred to the COVID ward on the same day as part of the infection control measures. Symptomatic treatment including anticipatory medications were prescribed. By that point, she deteriorated significantly and her clinical picture was consistent with Acute Hepatitis of unclear cause. Currently, as there are no approved therapies for COVID-19 infection despite all the supportive care, she sadly passed away on the 8th day of admission. Her liver screen was reported after her death which was negative for any acute or chronic identifiable causes of hepatitis.

Discussion

Liver dysfunction is more prevalent in cases of advanced COVID-19 as compared to the mild disease. A meta-analysis showed that liver functions were abnormal in 19% of patients as compared to the cases of less severe disease [4]. The liver injury mechanism in those patients is unknown. There can be several mechanisms involved, like direct damage to the hepatocytes from the virus, excessive inflammatory or immune related response, or hepatic and portal vein thrombosis as part of wide spread thromboembolic activities in those patients [5]. In a clinical setting, while treating the respiratory illness in the COVID-19 patients, chances of liver injury also requires equal attention. A good approach can be prescribing only those medications which can provide a protective effect for the liver functions and can also prevent the damages caused by inflammatory process resulting in a speedy recovery. However, we have very limited evidence to support this. According to the latest research, ACE II receptor is portal of entry for SARS-COV-2 and this receptor is present in the lung and the liver; [6] Nevertheless, the expression is much more higher in the cholangiocytes which is comparable to the pulmonary Type 2 Alveolar cells. This indicates that bile duct cells can be one of the important factor in the disease pathogensis [7]. Post mortem reports in COVID-19 patients also pointed towards drug induced damage in few cases with a list of theoretically approved medications like antibiotics and steroids [8,9].

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