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CASE REPORT

A Fulminant Hepatitis Case Triggered by COVID-19 Disease

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Özet

COVID-19 pandemisi Çin'in Wuhan kentinde, Aralık 2019'da ilk defa görülen akut respiratory syndrome coronavirus 2 (SARS- CoV-2), corona hastalığı 2019 (COVID-19) olarak bilinen, tüm dünyada milyarlarca insanı etkilemiştir. Başlarda yalnızca solunum yolu enfeksiyonu gibi görülse de, virüsle ilgili araştırmalar yapıldıkça gastrointestinal sistem tutulumu ve fekal oral yayılımın da önemli olduğu görülmüştür. Biz makalemizde; COVID-19'a bağlı fulminan seyreden bir hepatit vakasından bahsedeceğiz.

Anahtar Kelimeler: COVID-19, etilizm, hepatit.

Abstract

The COVID-19 pandemic, known as acute respiratory syndrome coronavirus 2 (SARS-CoV-2), corona disease 2019 (COVID-19), was seen for the first time in December 2019 in Wuhan, China, has affected billions of people all over the world. It has been seen that gastrointestinal system involvement and fecal-oral spread are also significant as researches on the virus are performed, however, it was initially seen as a respiratory tract infection. We will talk about a case of hepatitis with a fulminant course due to COVID-19 in our article.

Keywords: COVID-19, ethylism, hepatitis

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INTRODUCTION

Coronavirus disease (COVID-19), SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) betacoronavirus is a member of the genus family and is an enveloped, single-stranded RNA virus. It enters cells through the angiotensin-converting enzyme 2 (ACE-2) receptor (1). In many patients, gastrointestinal symptoms such as diarrhea, nausea-vomiting, and abdominal pain may accompany, however, the main symptoms are seen as fever, cough, and shortness of breath (2). It has been determined that the ACE-2 receptor is present in the gastrointestinal system besides the respiratory system (3). It has been shown that ACE-2 expression is detected in 2.6% of hepatocytes and 59.7% of cholangiocytes (4). The liver is the second most frequently involved organ after the lung (5). In a study, it was asserted that protein 7a specific to SARS-CoV-2 induces apoptosis in cell lines of the liver and organs in a caspase-dependent manner, and the possibility of SARS-CoV2 to directly affect liver tissue (6). Sonzogni et al. reported in an autopsy series of 48 cases that there were pathological liver findings suggestive of the focal portal–lobular lymphocytic infiltrates and hepatic vascular involvement (7).

CASE

A 30-year-old male patient applied to the emergency department of our hospital due to trauma. The patient was brought to our hospital's emergency service after he lost his balance and fell while driving under the influence of alcohol at a speed of 20 km/h. The patient has been drinking between 50-70 cc of alcohol per day for 10 years, the COVID-19 PCR (Polymerase Chain Reaction) test was positive 10 days ago, the rules of quarantine were applied at home for 10 days after he did not have any symptoms, and he did not take medication. The chest tomography taken was reported as normal, nothing remarkable is observed in the examinations. He followed the rules of home quarantine in this process. The patient applied to the internal medicine outpatient clinic in an external center with the complaint of not being able to lose weight 1 month before the emergency service application. In the examinations performed, nothing remarkable is observed except for Grade-2 hepatosteatosis in the whole abdominal ultrasound, and the fasting glucose test and lipid panel were within the normal range. In the patient's family; his mother has a history of congestive heart failure, and his father has a history of hypertension. His 2 sisters are alive and healthy. In the first physical examination of the patient in the emergency department, his general condition was moderate, he was conscious, his orientation and cooperation were normal, and his Glaskow coma scale was 15. Blood pressure was 130-70 mm/hg, pulse was 72/minute, oxygen saturation in room air was 93, and respiratory rate was 14/minute. Skin and sclera were icteric, convex appearance, mother-of-pearl striae were observed on abdominal examination, hepatomegaly, diffuse dullness was observed on palpation. Breath sounds were low at basal levels, no rales or rhonchi were heard. Heart sounds were heard deeply, no S1+, S2+ rubbing or murmur was heard. Pitting edema was observed in both lower extremities. In his examinations, glucose 80 mg/dL, urea 42 mg/dL, creatinine 0.8 mg/dL, Aspartate aminotransferase (AST) 72 U/L, Alanine aminotransferase (ALT) 356 U/L, total bilirubin 14 mg/dL, direct bilirubin 8 mg/dL, sodium 116 mEq/L , total protein 6.1 g/dL, albumin 3.0 g/dL, Hemoglobin 12 g/dL, MCV 83 fl, White

Blood Cell (WBC) 10.0 e3/uL, Platelet (PLT) 89.0 10e3/uL, International Normalized Ratio (INR) 4.2. Plasma ethyl alcohol level was negative. In the entire abdominal ultrasound, diffuse intra-abdominal fluid, liver dimensions of 170 mm, and spleen dimensions were measured as normal. On unenhanced chest tomography, bilateral pleural effusion reaching 3 cm was observed. The patient was admitted to the internal medicine service with acute hepatitis. The Child-Pugh score was calculated as C, Model for End-stage Liver Disease (MELD) score was calculated as 35. The hepatitis markers sent belonging to the patient were negative. Ammonia, autoimmune markers, viral markers, COVID-19 PCR tests were sent. Hypertonic fluid treatment was started. At the 4th hour of the follow-up, the patient had mental fog and a tendency to sleep. Control tests were sent and amino acid infusion treatment was started. INR increased to 9, sodium 116 mEq/L, and total bilirubin increased to 18.5 mg/dL in control examinations. The MELD score was recalculated as 41. The Glasgow coma scale of the patient decreased to 13, gastroenterology opinion was obtained for the patient, and fulminant hepatitis was accepted. Fresh frozen plasma was administrated. Since there was ethylism in the patient's history, first and second-degree relatives were questioned as priority donors. He was referred to the upper center where the transplantation was performed. In external center follow-ups, INR increased to 10, total bilirubin increased to 33 mg/dL. The patient died within 48 hours before transplantation. The COVID-19 PCR result sent on his admission was positive.

DISCUSSION

It is not known whether people with chronic liver disease are more susceptible to COVID-19 disease. During the COVID-19 infection, the presence of underlying chronic liver damage impairs the prognosis negatively and causes prolonged hospital stay (8). Not every increase in liver enzymes during COVID-19 infection may be due to the virus, and an increase is possible as a result of the use of hepatotoxic drugs used. For example; tocilizumab, acetaminophen, lopinavir/ritonavir, and remdesivir (9). Reported a case of drug-induced liver injury in a COVID-19 patient with a 40 times increase in transaminases after tocilizumab use. However, they stated that transaminases returned to normal after 10 days and suggested that the hepatotoxic effect may have been precipitated by the anti-viral agents used previously (10). On the other hand, since the ACE-2 receptor is in our intestines, liver, and biliary tract when the SARS-CoV-2 virus enters the cell, it should be expected that COVID-19 disease will cause complaints and clinical findings related to the digestive system and liver. While some of the patients may have severe liver damage characterized by jaundice, the frequency of mild liver damage detected by elevations of enzymes such as ALT, AST, and GGT without clinical symptoms varies between 10-50%. There may be a low level of bilirubin (which causes yellowing of the scleras and/or darkening of the urine color). It is mild liver damage and does not require any special treatment (11, 12). In recent studies, there are researches asserting that it is associated with worse outcomes in COVID-19 patients with a history of chronic liver disease. In a study, the mortality rate was reported as 40% in a research that includes

152 patients (including 103 patients with cirrhosis) with COVID-19 and chronic liver disease. In this study, patients; Child-Pugh (CP) classification was made, mortality rates were in order according to groups; A; reported as 23%, B 43%, and C 63%. While mortality in patients with cirrhosis was ascribed to pulmonary complications (79%), only causes related to liver disease were observed in 12% of patients (13, 14).

Conclusion: In patients hospitalized and followed up due to COVID-19 positivity, up to 2-3 times increase in liver enzymes can be observed. Our patient was asymptomatic at the time of diagnosis of COVID-19, and since there was no pathology in his examinations, he was included in the uncomplicated disease group, and our patient was not treated with any medication following the guideline (Republic of Turkey Ministry of Health General Directorate of Public Health adult patient treatment guide), and quarantine rules were applied at home for 10 days. However, in this process, the COVID-19 virus examined the development of fulminant hepatitis within days in our patient with hepatosteatosis. We tried to share this case because we thought we would contribute to the literature.

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