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LIVER FUNCTION COMPLICATIONS IN COVID-19 PATIENTS

Abstract.
The severe acute respiratory syndrome coronavirus 2 (SARS-Cov-2), the pathogen of 2019 novel coronavirus disease (COVID-19), has posed a serious threat to global public health. The World Health Organization (WHO) has declared the outbreak of SARS-CoV-2 infection an international public health emergency. Lung lesions have been considered as the major damage caused by SARS-CoV-2 infection. Current literature has many published clinical studies focusing on implications of hepatic involvement in COVID-19. However, most of them are diverse because of variation in definition of liver injury, different clinical presentations and severity of the disease in individual studies. Additionally, there is no strong evidence showing association of outcomes of COVID-19 in patients with pre-existing chronic liver disease or liver injury. Similarly, previous studies have shown that liver damage was common in the patients infected by the other two highly pathogenic coronavirus – severe acute respiratory syndrome coronavirus (SARS-CoV) and the Middle East respiratory syndrome coronavirus (MERS-CoV), and associated with the severity of diseases. In this review, the characteristics and mechanism of liver injury caused by SARS-CoV, MERS-CoV as well as SARS-CoV-2 infection were summarized, which may provide help for further studies on the liver injury of COVID-19.

Keywords: *COVID-19, liver, function, damage.*

Introduction. Coronavirus (CoVs) is a virus of the coronavirus family, which has the largest genome of all known RNA viruses and is widely found in humans, mice, pigs, cats, dogs and other animals. Seven coronavirus species are known to cause human disease, of which four species (HCoV-NL63, HCoV-229E, HCoV-OC43 and HCoV-HKU1) cause respiratory infections in immunocompromised individuals, infants and the elderly. The other three are highly pathogenic human coronaviruses, including the severe acute respiratory syndrome coronavirus (SARS-CoV), the Middle East respiratory syndrome coronavirus (MERS-CoV) and the 2019 new coronavirus (SARS-CoV-2). These three viruses can cause respiratory, intestinal, hepatic and neuronal diseases, and may lead to acute respiratory distress syndrome (ARDS), multiple organ failure (MOF) and even death in severe cases. Studies have shown that patients infected with SARS-CoV, MERS-CoV and SARS-CoV-2 may develop different degrees of liver injury. In this review, the characteristics and mechanism of liver injury caused by SARS-CoV-2 infection were summarized, which may provide help for further studies on the liver injury of COVID-19.

COVID-19 is caused by SARS-CoV-2, and typically manifests with systemic symptoms like fever and myalgia as well as respiratory symptoms including dry cough, dyspnoea and anosmia[4]. Reports suggest that lineage B b-coronaviruses that are highly pathogenic to humans such as the SARS-CoV (2002) and SARS-CoV-2 (2019) can affect the liver and induce acute hepatitis [2].

Recent studies on COVID-19 have shown that the incidence of liver injury ranged from 14.8% to 53%, mainly indicated by abnormal ALT/AST levels accompanied by slightly elevated bilirubin levels [1]. The albumin is decreased in severe cases and the level of albumin is around 26.3-30.9 g/L. The proportion of developing liver injury in severe COVID-19 patients was significantly higher than that in mild patients. In death cases of COVID-19, the incidence of liver injury might reach as high as 58.06% and 78%. One study reported that serum ALT and AST levels increased up to 7590 U/L and 1445 U/L, respectively, in a severe COVID-19 patient. Our unpublished data showed very similar findings to other studies, except that we found that serum GGT increased in severe cases and serum AKP level was at normal range in both mild and severe cases. Currently, studies on the mechanisms of SARS-CoV-2-related liver injury are limited. It has been shown that SARS-CoV-2 also uses ACE2 as its entry receptor as SARS-Cov does. Chai et al found that both liver cells and bile duct cells express ACE2 [5]. However, the ACE2 expression of bile duct cells is much higher than that of liver cells, but to a comparable level of alveolar type 2 cells in the lung. Bile duct epithelial cells are known to play important roles in liver regeneration and immune response. These results suggested that the liver injury occurred in COVID-19 patients may be due to the damage to bile duct cells, but not liver cells by the virus infection. Besides, the inflammatory cytokine storm was observed in severe COVID-19 cases, yet whether it results in liver damage in patients remains to be investigated. Postmortem biopsies were recently

performed in a death COVID-19 patient, and the results showed moderate microvascular steatosis and mild lobular and portal activity, indicating the injury could have been caused by either SARS-CoV-2 infection or drug-induced liver injury. Similar to the situation in SARS, antibiotics, antivirals and steroids are widely used for the treatment of COVID-19. These drugs are all potential causes of liver injury during COVID-19, but not yet being evident. Actually, a recent study reported that the liver injury observed in COVID-19 patients might be caused by lopinavir/litonavir, which is used as antivirals for the treatment of SARS-CoV-2 infection. So far, there is a lack of reports that liver failure occurs in COVID-19 patients with chronic liver diseases, such as chronic hepatitis B or C [6].

Conclusions. This review summarized the reports of liver injury caused by SARS-CoV-2 infection. The mechanisms of liver injury that occurred during SARS-CoV-2 infection remain largely unclear. Current understanding suggests that infection of highly pathogenic human coronavirus may result in liver injury by direct virus-induced cytopathic effects and/or immunopathology induced by overshooting inflammatory responses. Meanwhile, SARS-CoV may aggravate liver injury in patients with viral hepatitis, but there is no evidence for MERS-CoV and SARs-CoV-2. Importantly, drug-induced liver injury during the treatment of coronavirus infection should not be ignored and needs to be carefully investigated.

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СТОМАТОЛОГІЧНИЙ СТАТУС ДІТЕЙ З ЮВЕНІЛЬНИМ РЕВМАТОЇДНИМ АРТРИТОМ В ПРОЦЕСІ КОМПЛЕКСНОГО ЛІКУВАННЯ

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DENTAL STATUS OF CHILDREN WITH JUVENILE RHEUMATOID ARTHRITIS IN COMPLEX TREATMENT PROCESS

Анотація.

Отримані результати свідчать про достатньо ефективну дію запропонованого лікувально-профілактичного комплексу, який включав препарати імуномодулюючої, дезінтоксикаційної, протимікробної, антиоксидантної, мембраностабілізуючої та регулюючої мікробіоценоз дії, при стоматологічному лікуванні дітей 6-8 та 12-14 років з ювенільним ревматоїдним артритом. У дітей з даною патологією під дією лікувально-профілактичних заходів карієпрофілактична ефективність за 2 роки спостережень склала 40,7 % у 6-8 річних, а у 12-14 річних дітей – 45,1 %. Під дією лікувально-профілактичного комплексу