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LETTER TO THE EDITOR

Remdesivir and COVID-19

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To the editor:

The coronavirus SARS-CoV-2, first identified in November 2019 in China, is responsible for severe acute respiratory syndrome, the cause of the global pandemic COVID-19. To date, the global pandemic COVID-19 has caused about 1.04 million deaths and is currently ongoing [1, 2]. Several vaccines have entered phase 3 clinical trials. While waiting for effective vaccines, several antiviral drug treatments have been evaluated to treat the COVID-19 patient. To date, the data from epidemiological studies are not yet completely clear. Recent evidence associates some antiviral efficacy against SARS-CoV-2 with remdesivir [3]. Remdesivir is an antiviral drug of the nucleotide analogues family, which has been shown to have antiviral activity directed against several RNA viruses, such as Ebola virus, and coronaviruses responsible for MERS and SARS. It is currently considered a potential antiviral agent against SARS-CoV-2; in fact, in vitro studies have shown some activity to inhibit SARS-CoV-2. Several epidemiological studies are underway to evaluate the clinical antiviral SARS-CoV-2 efficacy of remdesivir. Recent evidence associates remdesivir with continuous and clinically significant improvements in COVID-19-positive patients, leading to a reduction in mortality and a decrease in recovery time [4]. While demonstrating some efficacy against SARS-CoV-2, the therapeutic treatment with remdesivir in COVID-19 patients requires additional data. Specifically, in the COVID-19 patient, remdesivir can be administered in combination with other drugs, increasing the risk of potential drug interactions. In fact, remdesivir is a substrate of enzymes such as CYP 3A4, CYP 2D6, and CYP 2C8 involved in the metabolism of several drugs used in the COVID-19 patient, and the potential co-administration of inhibitors may lead to a potential increase in its levels [5]. However, we believe that further aspects need to be considered and clarified, in particular the development of resistance to remdesivir by SARS-CoV-2. In addition, the severe COVID-19 patient may have extra pulmonary lesions, including liver, kidney, or cardiac lesions with development of arrhythmias [6–8]. A liver or kidney damaged by multisystem inflammation [9] caused by COVID-19 may require a modified and appropriate dose of remdesivir. Furthermore, the postmarketing data of remdesivir on the possible risk of long QT is unclear, and in a severe COVID-19 patient with cardiac lesions arrhythmias may be present, increasing the risk of cardiotoxicity. Epidemiological studies associate remdesivir with an efficacy against SARS-CoV-2, probably higher than other antivirals on the market, but further studies are needed to provide further data on this drug and on the treatment of COVID-19.

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Compliance with ethical standards

Competing interests The authors declare that they have no conflicts of interest.

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