Pharmacotherapeutics of SARS-CoV-2

Bajpai *et al.*[1] make a comprehensive review of Remdesivir, analyse current evidence and perspective in management of COVID-19 infection in May 2021 issue of the Journal. They scan through pivotal trials where safety and efficacy of this drug were tested in defined patient population, provide interaction profile and side effects assessment in their review.

Nevertheless, under a heading of Coronavirus pandemic – An update, the authors state that there are many potential candidates in the fray including chloroquine, hydroxychloroquine, ritonavir/lopinavir, plasma therapy, corticosteroids and interleukin hormones. "Many of these drugs", they go on, "have demonstrated antiviral activity previously and are hence being tried against this novel virus". Here what we want to emphasise is that corticosteroids are not antiviral but anti-inflammatory drugs. Recovery Collaborative Group writes that corticosteroids control inflammation when immunological process goes in hyperactive mode after first week of the COVID-19 illness.[2]

During first week of the illness when viral replication is going on at a fast pace, Remdesivir has value in that phase. When patient has a moderate disease, requires oxygen support to maintain his arterial oxygen saturation; then drugs inhibiting viral replication are demonstrated to provide clinical benefit in ACTT-1 trial. However, when patient enters second week, most of the times he clinically improves. Or in some small number of cases, his immunopathological response causes severe illness when his arterial oxygen saturation collapses. It's in these cases alone that immunosuppressive drugs are effective.

Making a distinction in this respect is important so that we prescribe this powerful drug suppressing our immunity only when it is indicated. But sad reality today is that due to failure to make this differentiation, these glucocorticoids are prescribed even outside this indication – resulting in an epidemic of Mucormycosis in our hospitals.[3,4]

Second, in the same paragraph, the authors state that in many countries case fatality ratio is more than 10%. What we need to realize here is that there is a need to make a distinction between infection fatality rate and case fatality rate and one should not compare one with another.[5] Moreover, the disease spread initially in early 2020 in nursing homes in Europe and United States and victims were elderly people with comorbidities. But later on, when young population got infected, mortality was much lower. Hence, we propose that the difference in case fatality is more a function of age and other demographic profile rather than geographic profile. This difference was taken into account so as to make priority groups for vaccination.

Another potential confounding factor is that in the first wave in the United States, Italy, United Kingdom, Spain, Brazil, Iran and several wealthiest parts of the world; hospitals got overwhelmed with the patients, many could not get bed inside, a few were treated in makeshift hospitals in camps on open grounds, and rest were left to die in their homes, in ambulances and at the gate of the hospitals.[6] Media widely reported that due to shortage of critical care beds, many seniors were said sorry, sent back to their homes and that has never occurred in this century or last one ever since the beginning the modern medical care system.

India saw this tormenting and unprecedented phenomenon in its second wave. Hence rather than some lethal variant or trait of the novel Coronavirus, this statistical figure may indicate capacity of healthcare system as well.

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Conflicts of interest

There are no conflicts of interest.

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We accessed all the webpages at the time of submission of this letter.

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