Pharmacotherapeutic Modifications in Cardiopulmonary Patients during COVID-19 Outbreak

Sir,

Severe acute respiratory syndrome coronavirus *(SARS-Cov-2)*, a human pathogenic virus now, has a well-established mechanism of host cell entry by angiotensin-converting enzyme-2 (ACE2 receptors) that are most commonly located on epithelial cells of the blood vessels, respiratory tract including lungs, intestine and kidney. Cardiopulmonary ailments are the most notable comorbidities for COVID-19 patients. Various recent studies conducted in Wuhan, China (the epicentre of COVID-19) have shown that an alarming number of 1,099 patients of polymerase chain reaction (PCR) – confirmed COVID-19 had severe diseases such as hypertension, diabetes mellitus of either type, coronary artery disease (CAD), renal and cerebrovascular diseases.[1]

Distinctively, the most consistent comorbidities described in various previous clinical studies in the victims of COVID-19 are commonly managed with angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs) adrenergic receptor blockers (β-blockers) and corticosteroids. In diabetic patients (types 1, 2) expression of ACE2 is significantly escalated, in those taking ACE inhibitors and ARBs.[2] Hypertension/CAD / hyperlipidemia / myocardial infarction prophylaxis and nonsteroidal anti-inflammatory drugs associated ulcers are also managed with ACE inhibitors and ARBs, which result in an upregulation of ACE2.[1] Medicines like thiazolidinediones (TZD), an antidiabetic agent and ibuprofen, a common painkiller can escalate ACE2 expression.[2] Agents that increase ACE-2 expression, increase the lethality index of COVID-19 by facilitating the host cell entry by the virus. Therefore, it is hypothesised that diabetes and hypertension treatment with ACE2-stimulating drugs escalates the possibility of developing acute and lethal COVID-19 infection. Pharmacologically, it is proven that ACE2 decreases inflammation and has been advocated as a prospective new therapy for inflammatory lung diseases, hypertension a combination of both ACE2 polymorphism and therapy. It is proposed that patients with diabetes, hypertension or any cardiac ailments, and those are receiving ACE2 stimulating agents for therapeutic purposes are at higher risk for acute COVID-19 infection. Hence, these should be replaced with other available options or monitored under strict medical supervision.

Respiratory chronic inflammation and infections are commonly treated with fluoroquinolones and corticosteroids. Fluoroquinolones increases the QT-interval and have a threat of evolving arrhythmias and exaggerated use of chloroquine or hydroxychloroquine in COVID-19; it expands the threat of developing arrhythmias; on the other hand, corticosteroids mask the immune response which results in more worsening the condition.[4] While β-blockers cause respiratory depression, which increase the severity in COVID-19 patients. Based on available pharmacological data, there is no study that suggests calcium channel blockers increased ACE2 expression. Therefore, calcium channel blockers can be a suitable alternative treatment in these patients.[5] Macrolides (Azithromycin) could be a better option than fluoroquinolones, as reported more efficacious in reducing secondary infections in COVID-19.[6] Paracetamol could be used rather than ibuprofen and diclofenac in COVID-19 patients.

It is suggested that by modifying pharmacotherapy, severity in cardiopulmonary patients during this COVID-19 outbreak can be reduced.

CONFLICT OF INTEREST:
Author declared no conflict of interest.

AUTHOR'S CONTRIBUTION:
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