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Antidepressant Drugs: Basic and Clinical Evidence and COVID-19

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Abstract

There is evidence to back up the idea that some antidepressants can lower levels of various cytokines in both humans and animals, and more recently, it has been revealed that some antidepressant have antiviral effect against SARS-CoV-2. The purpose of this narrative review is to assess the potential value of antidepressants in the management of COVID-19 infection as well as the potential advantages and disadvantages of antidepressant therapy for COVID-19 infections and mental problems. An analysis of the recent literature was done to determine the place of antidepressant drugs in the care of COVID-19 patients. Additionally, two phase II studies investigating fluvoxamine that were published and showed promising results against a placebo for clinical deterioration and hospitalisation rate. The first stages of seven ongoing clinical trials are examining fluvoxamine, fluoxetine and tramadol. The results from several observational studies, two phase II clinical testing, and the aggregate of the antiviral and anti-inflammatory laboratory development form the basis for ongoing clinical trials trying to investigate the potential use of antidepressants for COVID-19 infection in humans, despite the amount of evidence is limited.

Key words: COVID-19 • Tryptophan • pathological • Aspects • Serotonin

Introduction

The other two strains are Middle East respiratory syndrome and severe acute respiratory syndrome coronaviruses, both of which can cause fatal respiratory infections in people. SARS-CoV-2 was discovered to be a brand-new betacoronavirus that causes COVID-19, a disease that was proclaimed a pandemic in March 2020. Around the world, more than 543 million COVID-19 cases and 6,328,000 fatalities have been documented as of June 25, 2022. SARS-CoV-2 spreads via respiratory droplets and is influenced by a number of factors, including population density, the incidence of underlying diseases, and the availability of diagnostic testing. Age above 60, male sex, obesity, hypertension, diabetes, and chronic obstructive pulmonary disease are among the factors linked to illness severity that have identified. The SARS-CoV-2 spike protein's amino acid alterations were observed at the start of the pandemic. A strain with the substitution D614G was found, and it quickly overtook all other polymorphisms. The delta version of concern, which was initially discovered in India in December 2020, shares the G614 variation with the majority of circulating SARS-CoV-2 lineages. Until the Omicron version appeared, it then had the title of most likely to make globally. In November 2021, this type was discovered for the first time in Botswana and then quickly spread to South Africa. It spread quickly through numerous nations and was linked to an increase in infections. This variant differed from earlier versions in that it had more than 30 mutations in the spike protein, which are linked to improved transmissibility and enable the variant to more effectively evade infection- and vaccine-induced humoral immunity. Observational study findings have led to the current belief that Omicron may have a less severe clinical picture than other variations [1]. After the onset of COVID-19 symptoms, the median incubation period is four days, and the median time till the development of pneumonia is five days. The most frequent radiologic finding on chest computed tomography is ground-glass opacity. Leukopenia, thrombocytopenia, and lymphopenia are the most prevalent hematologic disorders at the time of admission. C-reactive protein and, less frequently, D-dimer, are both high in most individuals. Compared to patients with non-severe disease, patients with severe disease show more substantial laboratory abnormal values.

Numerous investigations on the clinical and pathological aspects of COVID-19's virological epidemic as well as studies on prognostic, predictive, clinical, and analytical factors have been published [2]. In individuals under 60 years old and with coexisting medical comorbidities, SARS-CoV-2 results in more severe clinical circumstances. Regarding the disparities between the sexes, male patients have a higher risk of getting serious illnesses and a higher mortality rate.

Discussion

The impact of COVID-19 infection in patients seeking mental health services has received little attention in published publications. Severe COVID-19 may be associated with mental problems. The risks of COVID-19-related death, hospitalisation, and intensive care unit admission in patients with pre-existing mental illnesses and exposure to various psychopharmacological medication classes were assessed using a thorough review and meta-analysis. 33 researches were included in the systematic review, and 23 papers were included in the meta-analysis. Additionally, we discovered two clinical trials that had already been published that demonstrated promising outcomes when fluvoxamine was administered versus a placebo in terms of clinical deterioration and the frequency of admission to an observation unit or hospitalized, respectively. Since the clinical trials found are continuing, there is no data from studies investigating tramadol and serotonin as possible COVID-19 therapies [3].

The potential positive action for COVID-19 infections is supported by two observational studies in addition to the clinical trial findings. With the prior use of any antidepressant as well as the use of SSRIs and SNRIs, a decrease in the composite variable of intubation or mortality was seen in one study. Escitalopram, fluoxetine, and venlafaxine are examples of SSRIs and SNRIs that have been shown to greatly reduce the need for intubation or cause mortality. In the other trial, antidepressant users showed a decreased risk of intubation or death. Antidepressant use was linked to favourable but not statistically significant outcomes. Findings regarding antiviral screening activity,

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epidemiological retrospective patient data, recent non-controlled series of cases, and two randomized placebo-controlled clinical trials evaluating the use of fluvoxamine in non-hospitalized patients, which showed promising positive results, all point to the possibility of repurposing antidepressants for the treatment of COVID-19. The objectives of this narrative review of the literature are to assess the potential contribution of antidepressants to the management of coronavirus infections and to determine the potential advantages and disadvantages of antidepressant therapy for patients with mental disorders and coronavirus infection. A recent in vitro investigation indicates that fluoxetine has antiviral effects on SARS-CoV-2, although these effects were not seen with paroxetine or escitalopram, other selective serotonin reuptake inhibitors.

Fluxetine isomers had comparable antiviral efficacy. Although the exact mechanism of action was unclear, fluoxetine led to a reduction in the expression of viral protein. Fluoxetine treatment decreased viral titers by up to 99%, pointing to a potential method involving direct functional inhibition of sphingomyelinase and impacting endolysosomal acidification. Another study found that fluoxetine had antiviral properties and suppressed Severe acute respiratory in vitro. In a polarization Calu-3 cell culture model, the combination of remdesivir and fluoxetine showed antiviral efficacy suppressing the formation of infectious SARS-CoV-2 nanoparticles by even more than 90%. When itraconazole and remdesivir were combined, similar outcomes were seen. In one published investigation, inhibitors of two coronavirus spike proteins were assessed [4]. These inhibitors were found by screening a library of licenced medications using tests for SARS-S and MERS-S pseudotyped particle entrance. Three library compounds were discovered by the authors to be broad-spectrum inhibitors of spike-mediated entrance using high-throughput screening methods.

Tryptophan metabolism and kynurenines have been found to be related to inflammation and immunity. In one study analysing the metabolome profile of 55 patients infected with SARS-CoV-2, the role of the tryptophan nicotinamide pathway seems to be clearly linked to inflammatory signals and microbiota, as well as to the possible involvement of cytosine, formerly described as a coordinator of cell metabolism in SARS-CoV-2. Interestingly, tryptophan represents a metabolic node that involves the synthesis of serotonin, the kynurenine pathway, and the indole/aryl hydrocarbon receptor pathway. Indole acetic acid is a ligand of AHR that has been linked to many disorders involving immune and inflammatory processes. Additionally, analysis of the serum from 33 COVID-19 patients and 19 negative controls revealed abnormal tryptophan metabolism in the kynurenine pathway, which seemed significantly control inflammation and immunity. According to a recent analysis on SSRIs, they may have a neuroprotective impact when exposed to COVID-19. Elevated levels both regional and systemic 5-HT may reduce the overproduction of proinflammatory cytokines. It has been suggested that selective serotonin re-uptake inhibitors, also known as results obtained drugs, may be used to treat COVID-19 because of their ability to affect endolysosomal trafficking. Sigma-1 receptor agonism, which various antidepressants, notably fluvoxamine, have showed, is another potential pathway for immunological modulation [5]. It has been demonstrated that the SSRI fluoxetine has antiviral effect by inhibiting endolysosomal acidification and cholesterol accumulation inside the endocytosis. Fluoxetine is also a functional inhibitor of sphingomyelinase. Accordingly, lysosome-targeting medicines have been proposed as a potential COVID-19 treatment based on preclinical findings. In most nations, the COVID-19 epidemic was accompanied by an increase in overall mortality rates at the national level. A higher risk of death is linked to a number of things.

Conclusion

Based on the antiviral and anti-inflammatory effects of various chemicals, some preliminary study hypothesises a potential benefit of several antidepressants. For the remaining ongoing clinical trials and potential future ones evaluating the experimental efficacy of using antidepressants for COVID-19 infection in humans, the findings from this antiviral and anti-inflammatory research, along with those from a few observational studies, two clinical trials, and other related research, serve as the foundation.

Conflict of Interest

None.

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