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Editorial

Pulmonary Arterial Hypertension and COVID-19

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Identification of the respiratory and cardiovascular systems, particularly, pulmonary arterial hypertension (PAH), acute cardiac injury, and venous thromboembolism (VTE) can be identified in long-term effects of COVID-19, but the virus also affects the neurological system, bones, endocrine glands, include acute respiratory distress syndrome (ARDS) [1,2].

With a novel, and severe novel coronavirus infections, PAH has been reported to complicate the course of illness for 13.4% and 21% of patients, respectively. PAH is a serious complication of new coronavirus infection, increasing the likelihood of requiring mechanical ventilation, extracorporeal membrane oxygenation (ECMO), intensive care unit (ICU) care and even death. Enhancing the long-term prognosis of patients and minimize the hospitalization rate and death due to such complications therefore, by early detecting high pulmonary artery pressure in patients with SARS-CoV-2 [3-6]. As revealed by previous studies and reasons described by the processes of immunological dysfunction, endothelial dysfunction, vascular leakage, and thrombotic microangiopathy that are comparable to those that cause pulmonary vascular disease, such as PAH may be responsible for the effects of SARS-CoV-2 on pulmonary hemodynamics. On the other hand, reports of the study mechanism's depth and specificity are uncommon [3-7].

A recent study in 2024 conducted by Hou., et al. using a functional enrichment analysis on the GEO database to identify common differentially expressed genes (C-DEGs) across the PAH and COVID-19 datasets. The results of a screening of the key genes using three machine algorithms: LASSO, RF, and SVM-RFE-based were confirmed by the validation queue. The role of prioritized core genes was examined by using the gene set enrichment analysis (GSEA). The regulatory networks including these DEGs, including TF-gene connections and TF-microRNA co-regulation were next mapped out. Molecular docking simulations, drug-protein interaction networks, and molecular dynamics simulations were employed to screen for possible therapeutic medicines. The study findings are expected to offer a novel approach to elucidating the genetic connection between the aforementioned disorders (Figures 1-5) [8].

Additionally, CCL20 and SELE were found to be indicators of PAH and COVID-19 co-pathogenesis by various bioinformatics analyze and machine learning algorithms. Adaptive immune response, leukocyte, lymphocyte mediated immune responses, and proinflammatory response mediated by cytokines like IL-12, TNF-α were functionally enriched in these two hub genes. These two hub genes were selected for nomogram construction and their diagnostic value evaluated by machine learning. The nomogram was found to have high diagnostic value. Dendritic cells had the strongest connection with CCL20 and SELE, followed by activated CD4 T cells, active dendritic cells, natural killer T cells, neutrophils, and plasmacytoid dendritic cells. Using only 2 reference genes, they were able to isolate 12 shared TFs and 25 shared TF-miRNAs.by FFL tool, This FFL among CCL20, miR-1256 and PPARG may be a novel regulatory module in PAH complicated with COVID-19. It was hypothesized that AFLATOXIN B1, 1-NITROPYRENR, and FENRETINIDE would

be useful in treating PAH complicated with COVID-19. Further molecular dynamics and molecular docking simulations demonstrated that 1-nitropyrene had the most stable binding with CCL20 and SELE (Figures 1-5) [8].

In conclusion, by understanding the comorbidity of PAH and COVID-19 may be assisted by these angiogenesis and biomarkers connection between PAH and COVID-19.

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