First assignment

Bioinformatics and system biology approach to identify the influences of COVID-19 on cardiovascular and hypertensive comorbidities

INTRODUCTION

Recent studies have tracked down that some prior conditions significantly increment the danger of extreme side effects and mortality in COVID-19 patients. These contain pulmonary diseases, cardiovascular diseases, kidney disease, type 2 diabetes, and hypertension. While COVID-19 essentially influences the respiratory system in the beginning stages of the disease, while it additionally influences the cardiovascular system of patients, extraordinarily increasing the risk of fatality. Numerous examinations revealed raised death rates in patients with SARS-CoV-2 infections and increased levels of markers of chronic heart failure. Also discovered hypertension as comorbidity is related with an increased danger of extreme disease with SARS-CoV-2 disease. discovered 32% of COVID-19 patients with another medical issue. studies are needed to identify the interactions between these diseases and COVID-19. Further, the treatments under the examination for COVID-19 may affect cardiovascular disease, hypertension, or other critical comorbidities by impacting the pathways that intercede their interaction with COVID-19.

Angiotensin-Converting Enzyme 2 (ACE2) is known to play an important role in facilitating SARS-CoV-2 cell entry, and this gene is accounted for many human tissues including the intestine, testis, kidneys, heart, and lungs. ACE usually converts Angiotensin I in the renin-angiotensin-aldosterone system (RAAS) of human physiology to Angiotensin II that impact human blood pressure. ACE2 interacts with SARS-CoV2 that may cause vascular dysfunction. ACE inhibitors is used as treatments for hypertension and cardiovascular disease by modulating the RAAS pathway. These drugs inhibit the RAAS and interrupt the performance of ACE and increase the level of ACE2 receptors. In any case, recent investigations found that increased ACE2 levels can be gainful in COVID-19, This serves to show the possible significance of interaction among cardiovascular diseases and hypertension with COVID-19.

In this paper, we considered the interaction of COVID-19 with chronic heart failure (CHF) and idiopathic portal hypertension (IPH), pulmonary arterial hypertension (PAH), and preeclampsia.

CHF is a condition that reduces cardiac output and blood oxygenation insufficiency. **IPH** is a condition of blood pressure in the portal vein and its branches. **PAH** is a case of increased blood pressure within the lungs. **Preeclampsia** is a pregnancy complication that is characterized by hypertensive conditions.

We have found significant cell signaling pathways and gene networks that are commonly associated with these diseases and SARS-CoV-2 infection on human blood cells. The identified pathways and networks are associated with other diseases that may lead to improved therapeutic way for life-threatening SARS-CoV-2 infections.

ABSTRACT

Severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) infected persons that have hypertension or cardiovascular comorbidities have an elevated risk of serious (COVID-19) disease and a high rates of mortality but how COVID-1919 and cardiovascular diseases interact are obscure. We looked to identify a new mechanism of interaction by recognizing genes with expression in SARS-CoV-22 infection that is relevant to the pathogenesis of cardiovascular disease and hypertension. Some research shows the SARS-CoV-22 uses the angiotensin-converting enzyme-2 as a receptor to infect human susceptible cells.ACE2 usually converts Angiotensin I in the renin-angiotensin-aldosterone system to Angiotensin II, which affects blood pressure levels. We use bioinformatics to identify genetic links using MRNA data peripheral blood cells from COVID-19 patients and compared them with blood samples from patients with either chronic heart failure disease or hypertensive diseases. Differentially expressed genes (DEGs) popular to COVID-19 and chronic heart failure, and common to COVID-1919 and hypertension, COVID-1919 does not share a large number of differentially expressed genes with the conditions under consideration. However, those that were recognized included genes playing roles in T cell functions, such as receptor pathways, cytokines, chemokines, cell stress, type 2 diabetes, and gastric cancer. The result of this study may help in recognizing significant targets of treatment that can combat the progressing pandemic due to SARS-CoV-22 infection.

RELATED WORK

1-Md MA, Aktar S, Md R-A-M, et al. A machine learning model to identify early stage symptoms of sars-cov-2 infected patients. Expert Sys Appl 2020; 160:113661. **2-**Aktar S, Talukder A, Ahamad M, et al. Machine learning and meta-analysis approach to identify patient comorbidities and symptoms

that increased risk of mortality in covid-19. arXiv preprint arXiv:200812683 2020. **3-**Alexander MR, Norlander AE, Elijovich F, et al. Human monocyte transcriptional profiling identifies il-18 receptor accessory protein and lactoferrin as novel immune targets in hypertension. Br J Pharmacol 2019; 176(12): 2015–27. **4-**Badesch DB, Champion HC, Sanchez MAG, et al. Diagnosis and assessment of pulmonary arterial hypertension. J Am Coll Cardiol 2009; 54(1 Supplement): S55–66. **5-**Barrett T, Wilhite SE, Ledoux P, et al. NCBI GEO: archive for functional genomics data sets–update. Nucleic Acids Res 2012; 41(D1): D991–5 11. **6-**Chowdhury UF, Shohan MUS, Hoque KI, et al. A computational approach to design potential sirna molecules as a prospective tool for silencing nucleocapsid phosphoprotein and surface glycoprotein gene of sars-cov-2. bioRxiv 2020. **7-**Coperchini F, Chiovato L, Croce L, et al. **9-**The cytokine storm in covid-19: an overview of the involvement of the chemokine/chemokine-receptor system. Cytokine Growth Factor Rev 2020.8-Diriba K, Awulachew E, Getu E. The effect of coronavirus infection (sars-cov-2, mers-cov, and sars-cov) during pregnancy and the possibility of vertical maternal–fetal transmission: a systematic review and meta-analysis. Eur J Med Res 2020; 25(1): 1–14. **10-**Ferrario CM, Jessup J, Chappell MC, et al. Effect of angiotensin-converting enzyme inhibition and angiotensin ii receptor blockers on cardiac angiotensin-converting enzyme 2. Circulation 2005; 111(20): 2605–10. **11-**Fornes O, Castro-Mondragon JA, Khan A, et al. JASPAR 2020: update of the open-access database of transcription factor binding profiles. Nucleic Acids Res 2019; 48(D1): D87–92 **12-**Guo J, Huang Z, Lin L, et al. Coronavirus disease 2019 (covid-19) and cardiovascular disease: a viewpoint on the potential influence of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers on onset and severity of severe acute respiratory syndrome coronavirus 2 infection. J Am Heart Assoc 2020; 9(7):e016219. **13-**Gurwitz D. Angiotensin receptor blockers as tentative sars-cov-2 therapeutics. Drug Dev Res 2020. **14-**Guzik TJ, Mohiddin SA, Dimarco A, et al. Covid-19 and the cardiovascular system: implications for risk assessment, diagnosis, and treatment options. Cardiovasc Res 2020. **15-**Md NH, Babul Islam M, Chowdhury UN, et al. Network-based computational approach to identify genetic links between cardiomyopathy and its risk factors. IET Syst Biol 2020; 14(2): 75–84. **16-**Hamacher J, Sadallah S, Schifferli JA, et al. Soluble complement receptor type 1 (cd35) in bronchoalveolar lavage of inflammatory lung diseases. Eur Respir J 1998; 11(1): 112–9. **17-**Hernández-Gea V, Baiges A, Turon F, et al. Idiopathic portal hypertension. Hepatology 2018; 68(6): 2413–23. **18-**Md EH, Uddin S, Khan A, et al. A framework to understand the progression of cardiovascular disease for type 2 diabetes mellitus patients using a network approach. Int J Environ Res Public Health 2020; 17(2): 596. **19-**Ishiyama Y, Gallagher PE, Averill DB, et al. Upregulation of angiotensin-converting enzyme 2 after myocardial infarction by blockade of angiotensin ii receptors. Hypertension 2004; 43(5): 970–6.**20-**Lee CR, North KE, Bray MS, et al. Cyclooxygenase polymorphisms and risk of cardiovascular events: the atherosclerosis risk in communities (aric) study. Clin Pharmacol Therap 2008; 83(1): 52–60.