Vol 5 | Issue 1 | Pages 130-131

Archives of Family Medicine and General Practice

ISSN: 2578-6539

Letter to Editor

DOI: 10.36959/577/494

Letter to the Editor: Host Response to SARS COV-2 and its Effect

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SARS COV-2 is an airborne disease. Clinical and lab appearances incorporate fever, chills, myalgia, running nose, dyspnoea, pneumonia, lymphopenia, neutrophilia, thrombocytopenia, and raised serum lactate dehydrogenase, alanine aminotransferase, and creatine kinase. Medical service providers are at the highest risk and the aged people with multiple comorbid diseases are also vulnerable. The treatment has been exact, and there is no authorized SARS COV-2 vaccine for people up until now. Notwithstanding, the nearness of enduring killing antibodies and memory T-and B-lymphocytes in improving SARS patients raises trust in dynamic inoculation. Moreover, results from preclinical SARS immunizations communicating spike protein to inspire killing antibodies and cell reactions that are defensive in mouse and nonhuman primate models are empowering.

Data from other free examinations moreover show that SARS-express T lymphocytes against S, M, E, and N proteins are recognized in recovering SARS tests from one to four years post-infection using covering peptides against singular fundamental proteins, rather than a genome-wide system [1]. All proteins are IFN- γ positive and both CD4 and CD8 responses are distinguished for it. Without antigen and reexposure, memory T cells express to SARS-CoV further declined and could be perceived in a little degree of picking up quality patients [2]. Using a leading group of TCR-unequivocal antibodies, effector/memory Vγ9Vδ2 cells were found in recuperating SARS patients [3].

These cells are connected with a higher adversary of SARS IgG levels. Fortified Vγ9Vδ2 cells show IFN-γ-subordinate adversary op SARS-CoV development and can kill SARS-CoV debased target cells. Additionally, using a desire figuring to perceive putative cytotoxic T-lymphocyte (CTL) epitopes, S-express CTL in mending SARS patients show an isolated effector phenotype, which is depicted by CD45RA+CCR7−CD62L−CCR5+CD44+ [4]. In an alternate report, the CTL epitopes perceived could be used to induce CTL responses in A2 transgenic mice after DNA inoculation [5]. Numerous infectious diseases have been associated with HLA gene polymorphisms. The relationship of express HLA alleles with feebleness to SARS pollution and disease reality has been proposed [6]. Regardless, as a result of various polymorphism of HLA alleles and the unobtrusive number of tests attempted, the results are questionable. The human genome HLA area has been recognized for its relevance for both disease risk and resistance [7].

In mild and extreme cases of COVID-19, differential immune responses have been reported, including delayed IgM responses and higher S protein IgG titers in non-ICU patients [8,9]. A recent study of a SARS-CoV-2 genome-wide SNP interaction study using viral genomes of full length detected an SNP associated with COVID-19 intensity at nucleotide 11083 [10]. The response to SARS-CoV-2 is imbalanced about regulating virus replication versus activation of the adaptive immune response. This SNP is located in the non-structural nsp6 protein [10]. COVID-19 therapies have less to do with the response of the IFN and more to do with inflammation management.

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Accepted: November 10, 2020

Published online: November 12, 2020

Citation: Rahman S, Rahman M, Sarwar ASM, et al. (2020) Letter to the Editor: Host Response to SARS COV-2 and its Effect. Arch Fam Med Gen Pract 5(1):130-131

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DOI: 10.36959/577/494