

## Letter to the Editor

## Can COVID-19 Be a Risk Factor for Tuberculosis?

Benyamin Parseh<sup>1</sup>, Ehsan Allah Kalteh<sup>2\*</sup>, Mahnaz Sheikhi<sup>3</sup>, Mousa Ghelichi-Ghojogh<sup>4</sup>

1. Ph.D., Applied Cell Sciences, Faculty of Advanced Medical Technologies, Golestan University of Medical Sciences, Gorgan, Iran
2. MSc of Epidemiology, Infectious Disease Research Center, Golestan University of Medical Sciences, Gorgan, Iran
3. General Practitioner, Golestan University of Medical Sciences, Gorgan, Iran
4. Ph.D. Candidate of Epidemiology, Student Research Committee, Shiraz University of Medical Sciences, Shiraz, Iran.

**\*Corresponding Author:** Ehsan Allah Kalteh, MSc of Epidemiology, Infectious Disease Research Center, Golestan University of Medical Sciences, Gorgan, Iran. Email: [kalteh270@yahoo.com](mailto:kalteh270@yahoo.com)

**Please cite this article as:** Parseh B, Kalteh EA, Sheikhi M, Ghelichi-Ghojogh M. Can COVID-19 Be a Risk Factor for Tuberculosis? J Cell Mol Anesth. 2021;6(1):104-5. DOI: <https://doi.org/10.22037/jcma.v6i1.32694>

## Dear Editor

Tuberculosis is a chronic bacterial disease often caused by mycobacterium tuberculosis(1). In 2017, 10 million people were infected to and 1.6 million people died of tuberculosis (TB) (2). In patients with pulmonary TB, infectious aerosols are distributed in the air and enter other's lungs in inspiration. It passes the mucociliary defense line and enters the pulmonary terminal alveolus where it proliferates and may get disseminated to the whole body through blood circulation. Delayed allergic reactions and cellular immunity are formed 4 to 8 weeks after infection. In 90-95% of patients, cellular immunity manages to inhibit bacillus proliferation. Though, several bacilli may persist and get reactivated in immunocompromised patients within months to years and cause re-infection and active TB (1, 3). Nearly, a third of the world's population is carrying latent TB. Immune system suppression in patients suffering from HIV, diabetes, malnutrition, smoking, alcohol abuse, opium addiction, and immunosuppressive drugs, increases the risk of tuberculosis disease in patients with latent TB infection (3).

According to the latest statistics published by WHO, a total of 42,512,186 approved COVID-19 cases and 1,147,301 deaths have been reported since the first case report in Wuhan on October 25, 2020 (4). The demolishing consequences of viral infections are

mainly caused due to uncontrolled excess release of proinflammatory cytokines. Proinflammatory cytokine overexpression is stimulated through different pathways including overexpression of Toll-Like Receptors (TLR) (5). Most COVID-19 severe cases are presented with an uncontrolled elevated level of proinflammatory cytokines such as IL-6, IL-1 $\beta$ , IL-2 IL-8, IL-17, G-CSF, GM-CSF, IP10, MCP1, CCL3, and TNF which is called a cytokine storm. An elevated level of proinflammatory cytokines may lead to shock and damage to different organs including the heart, kidney, liver, and respiratory system (6, 7). In terms of pathophysiology, TB is an unresolved inflammation caused by the host's inability to remove the pathogen. Both pro-inflammatory and anti-inflammatory pathways are activated and deactivated in TB (8). Accordingly, the response varies based on the disease stage and the involved organ. This imbalance, rather than a severe inflammatory response is the characteristic feature of TB (5). Previous studies showed that proinflammatory cytokine overexpression such as TNF and IL-6 exacerbates TB (9, 10). In COVID-19 patients, we are facing immune system imbalance due to cytokine storm. Thus, COVID-19 may be a predisposing factor to turn latent TB into active TB. If COVID-19 acts as a risk factor for TB occurrence, the COVID-19 burden will prevent form TB global elimination. Since the effect of COVID-19 on TB is not yet investigated, it is suggested to evaluate

the relationship between COVID-19 and active TB risk in future studies.

## Conflicts of Interest

The authors declare that there are no conflicts of interest.

## References

1. Pareek M, Greenaway C, Noori T, Munoz J, Zenner D. The impact of migration on tuberculosis epidemiology and control in high-income countries: a review. *BMC medicine*. 2016;14:48.
2. Mohammed H, Oljira L, Roba KT, Ngadaya E, Ajeme T, Haile T, et al. Burden of tuberculosis and challenges related to screening and diagnosis in Ethiopia. *J Clin Tuberc Other Mycobact Dis*. 2020;19:100158.
3. MacNeil A, Glazier P, Sismanidis C, Maloney S, Floyd K. Global Epidemiology of Tuberculosis and Progress Toward Achieving Global Targets - 2017. *MMWR Morb Mortal Wkly Rep*. 2019;68(11):263-6.
4. Florez H, Singh S. Online dashboard and data analysis approach for assessing COVID-19 case and death data. *F1000Res*. 2020;9:570.
5. Kaufmann SHE, Dorhoi A, Hotchkiss RS, Bartenschlager R. Host-directed therapies for bacterial and viral infections. *Nat Rev Drug Discov*. 2018;17(1):35-56.
6. Rajaei S, Dabbagh A. The immunologic basis of COVID-19: a clinical approach. *J Cell Mol Anesth*. 2020;5(1):37-42.
7. Xu Z, Shi L, Wang Y, Zhang J, Huang L, Zhang C, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. *Lancet Respir Med*. 2020;8(4):420-2.
8. Etna MP, Giacomini E, Severa M, Coccia EM. Pro- and anti-inflammatory cytokines in tuberculosis: a two-edged sword in TB pathogenesis. *Semin Immunol*. 2014;26(6):543-51.
9. Dorhoi A, Kaufmann SH. Tumor necrosis factor alpha in mycobacterial infection. *Semin Immunol*. 2014;26(3):203-9.
10. Casarini M, Ameglio F, Alemanno L, Zangrilli P, Mattia P, Paone G, et al. Cytokine levels correlate with a radiologic score in active pulmonary tuberculosis. *Am J Respir Crit Care Med*. 1999;159(1):143-8.