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Journal of Human Virology & Retrovirology

Editorial

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Pathogenesis and pulmonary pathology of covid-19

Editorial

In late December 2019, a first-reported cluster of patients with unknown-etiology pneumonia was admitted to hospitals in Wuhan city, China. The onset of this potential coronavirus outbreak provided the estimate of a reproduction number for 2019 novel coronavirus (named by the World Health Organization "COVID-19" on February 11, 2020) that was significantly larger than 1 (ranges from 2.24 to 3.58). These admitted patients were epidemiologically associated with a wet animal and seafood wholesale market in Wuhan city, Hubei province, China. The incubation period of COVID-19 infection is around 5.2 days. As of January 22, 2020, the first 17 deaths of COVID-19 with the median age of 75 (range 48-79) years was reported by the China National Health Commission.

COVID-19-infected patients demonstrated higher numbers of leukocytes, increased significantly plasma levels of pro-inflammatory cytokines, high erythrocyte sedimentation rate (ESR) and D-dimer, Interleukin (IL)-1-β, IL-1-RA, IL-7, IL-8, IL-9, IL-10, basic Fibroblast Growth Factor (FGF)-2, Granulocyte-Colony-Stimulating Factor (GCSF), Granulocyte-Macrophage Colony-Stimulating Factor (GMCSF), Interferon (IFN)-γ, Interferon-γ-induced Protein (IP)-10, Monocyte Chemoattractant Protein (MCP)-1, Macrophage Inflammatory Protein (MIP)-1-α, MIP-1-β, Platelet-Derived Growth Factor (PDGF)-B, Tumor necrosis factor (TNF)-α, and Vascular Endothelial Growth Factor (VEGF)-A. Some severe COVID-19 cases with intensive care unit admission demonstrated high levels of IL-2, IL-7, IL-10, GCSF, IP-10, MCP-1, MIP-1-α, and TNF-α. The main pathogenesis of COVID-19 infection was severe pneumonia, RNAaemia, ground-glass opacities on the chest roentgenogram, and acute cardiac injury. Nevertheless, one of the COVID-19 case reports revealed leukopenia (70 % were neutrophils) and high level of C-reactive protein at 5 days of fever (39.0°C) with presence of a cough and coarse breathing sounds of both lungs. Pathologic pulmonary examination of two patients with adenocarcinoma and COVID-19 infection revealed reactive hyperplasia of pneumocytes, particularly type II pneumocytes with patchy inflammatory cellular infiltration and multinucleated giant cell infiltration, fibroblastic plugs in air spaces, proteinaceous exudate, and pulmonary edema.

In conclusion, the report of histopathologic pulmonary findings contributing to better understanding of the mechanism that COVID-19 causes lung injury in the patients in Wuhan city, China and worldwide.

Volume 8 Issue 1 - 2020

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Received: April 06, 2020 | **Published:** April 27, 2020

Acknowledgments

None.

Conflicts of interest

The author declares no conflict of interest.