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Editorial

Antibody Response and Viral Load in COVID-19 Severity

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Patients with various severities of COVID-19 (coronavirus disease-2019) demonstrates different viral shedding patterns and antibody responses [1]. Due to finding of IgM in tissues outside the respiratory tract in severe COVID-19 patients, detection of urinary and other body fluid antibody responses could be used as a biomarker to determine disease severity [1]. Strong cross-reactivities were detected between SARS-CoV-2 (COVID-19) and SARS-CoV, but not MERS-CoV (middle-east-respiratory-syndrome coronavirus) that is significant information for the differential diagnosis [1]. In comparison to mildly ill patients, severely ill patients have more prolonged viral shedding in various tissues and have more IgM response [1]. A recent study among 94 patients with COVID-19 from the Guangzhou Eight People's Hospital, China demonstrated that SARS-CoV-2 (severe acute respiratory sundrome-coronavirus-2) (COVID-19) viral load (VL) peaked at 0.7 days before the symptom onset, whereas SARS-CoV VL peaks on average 10 days after the onset of symptoms [2-5]. There is no different of viral load kinetics between mild and severe COVID-19 patients [5]. Similarly, a pervious study among 5,000 COVID-19 patients from Lombardy, Italy revealed no VL difference between asymptomatic carriers and symptomatic patients [6], whereas a previous study among 76 COVID-19 patients in Nangchang, China demonstrated that hospitalized severe-COVID-19 patients tend to have a high VL and a longer virus-shedding periods, in comparison to mild patients [7]. Liu., *et al*. demonstrated that mild COVID-19 patients had significantly lower VLs compared with severe patients [7]. Wölfel., *et al*. revealed that COVID-19 patients had upper respiratory VL peaks within the first week of symptoms [8]. Patients in this study continued to have active viral replication in upper respiratory tract tissue detected by PCR despite 100 % seroconversion of the patient cohort by day 14 and symptom cessation [8]. Severe COVID-19 patients had significant prolonged symptomatic duration [8]. ICU patients remained PCR positive with a prolonged symptom duration, compared with non-ICU patients [9]. Severe symptoms in COVID-19 patients are likely not associated with high viral titers [10]. Acute respiratory distress syndrome (ARDS), multiple organ failure, related-immunologic hyperactivation (high level of various cytokines, like interleukin (IL)-6, tumor necrosis factor (TNF)-alpha, lymphocyte activation, T-helper 17 differentiation, severe lymphopenia) seems to be associated with patient deterioration [11,12].

Technology-based-polymerase-chain reaction (PCR) allows calculation of VL that is associated with transmission risk and viral disease severity [13]. Pujadas., *et al*. recently demonstrated that there was an independent association between high VL and the mortality of 1,145 COVID-19 patients (hazard ratio 1.07 (95% Confidential Interval (CI) 1.03 - 1.11, p (probability) = 0.0014, Cox proportional hazards model adjusting for age, sex, asthma, atrial fibrillation, coronary artery disease, chronic kidney disease, chronic obstructive pulmonary disease, diabetes, heart failure, hypertension, stroke, and race), with a 7% increase in hazard for each log transformed copy per millimeter (ml) [14]. By univariate survival analysis, the study demonstrated that there was a significantly statistical difference in survival probability between those with low VL (p = 0.0003) and those with high VL (greater than the overall mean log10 VL of 5.6 copies per ml), with a maximum follow-up of 67 days and a mean follow-up of 13 days (standard deviation (SD) 11) [14]. VL might affect isolation measures on the basis of infectivity [14]. Nevertheless, no current actual studies have evaluated the association between VL and mortality in large patient cohort [15-17]. Antibody responses against N or S protein of SARS-CoV-2 (COVID-19) are associated with neutralizing antibody titers that may be useful for passive transfusion therapy in COVID-19 [1].

In conclusion, further urgent studies should be identification of the parameters associated between the viral load and clinical parameters, such as certain comorbidities, symptom severity, hospital admission and direct hospital discharge, hospital length of stay, intensive-care-unit (ICU) admission, length of need for oxygen support, and overall survival. Further exploration quantitative VLs from lower respiratory tract tissue and blood in severe COVID-19 patients may prove to be a better predictor for clinical outcomes. Future studies will address SARS-CoV-2 (COVID-19) VL dynamics and the quantitative association with neutralizing antibodies, cytokines, pre-existing conditions and therapies.

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