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Journal of Infection 82 (2021) e35

Contents lists available at ScienceDirect

Journal of Infection

journal homepage: www.elsevier.com/locate/jinf

Letter to the Editor

SARS CoV-2 viral load might not be the right predictor of COVID-19 mortality ☆

In their recent correspondence, Pujadas et al. have reported SARS-CoV-2 viral load at diagnosis as an independent predictor of mortality. They've reported the Mean log10 viral load significantly differed between patients who were alive (n=807; mean log10 viral load 5•2 copies per mL [SD 3]) versus those who had died (n=338; 6•4 copies per mL [2•7]) by the end of the study period.[1] The author would like to argue the clinical significance of their findings as it's apparent from the standard deviation of their reported results that some patients have survived though their initial detected viral load has been like or above the mean viral load in the deceased group and vice versa. Noteworthy, a systematic review of literature has previously demonstrated that seven studies observed increases in SARS CoV-2 viral loads prior to clinical deterioration and vice versa, yet it's also reported other seven studies that found little to no difference in viral load between presymptomatic, asymptomatic and symptomatic patients.[2] In a trial to explain the apparent contradictory results found in different studies as well as the lack of a distinct boundary between the viral loads that might be associated with a higher mortality rate or a higher recovery rate; the author would like to suggest that SARS CoV-2 viral load should be only considered as a personalized reflection of the immune response to COVID-19 as well as of the genetic polymorphisms in SARS CoV-2 receptors.[3] ACE2 polymorphisms might be a better field of study than SARS CoV-2 viral load wishing to develop a genetic test that might predict and exempt, if possible, from COVID-19 related duty those who are more vulnerable to complications and mortality.[4]

Declaration of Competing Interest

None.

Funding/financial disclosure

None

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https://doi.org/10.1016/j.jinf.2020.08.018
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