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Editorial

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Having to live with COVID-19 infection: an increasingly, concrete risk

Marco Bongiovanni*

Pneumology Unit, Department of Medicine, Ospedale Salvini, Garbagnate Milanese, ASST Rhodense, Milan, Italy

Introduction

Many factors contributes to the transformation of the coronavirus disease (COVID-19) from an endemic to a pandemic [1]. SARS-CoV-2 showed to be highly contagious with both droplets and contact transmission, leading to a huge spread in the number of infected patients; the extremely variable period of incubation (2.4-15.5 days) can further elicit this process [2]. As a consequence, the human to human transmission plays a crucial role in disease spread. At the very beginning of the pandemic, it almost took 1,5 month that the outbreak was controlled in China, with a primary increasing trend, steep slope, and finally the downtrend. This demonstrates not only the normal trend of an infectious outbreak but also the role of human intervention to control it. The beginning of the uptrend in other country was almost simultaneous with the beginning of the downtrend in China; this can be explained by the role of incubation period in estimating the true prevalence of the disease. Although many patients were potentially infected in other countries, they were asymptomatic carriers in the incubation period and could silently spread the infection to other susceptible individuals. Furthermore, at the beginning of the pandemic some diagnostic bias could have led to underestimate the real prevalence of the disease; as the symptoms of COVID-19 were similar to other pneumonias, some of the mild to moderate cases could have been clinically diagnosed as community acquired or viral pneumonia without being tested for COVID until late stages. Overall, after infecting over 118,000 patients in over 100 countries, and causing more than 4200 deaths, on March 11th 2020 the WHO declared that the COVID-19 outbreak was a global pandemic.

Some assumptions formulated at the beginning of the pandemic on COVID-19 widespread showed to be false or, at least, questionable. One assumption was that SARS-CoV-2, similar to H1N1 virus, will attenuate with high temperature, while we have already observed a second wave of the pandemic in some countries, despite rising temperatures. Another assumption was reaching herd immunity for SARS-CoV2 as the number of the affected individuals would rise, thus theoretically restrict the transmission of virus due to the increased number of immune host and to the decreased number of susceptible ones [3]. However, a key concept to develop and maintain herd immunity is a strong immunization against the pathogens which lasts long enough so that the fraction of the population immune to the virus surpasses the herd immunity threshold. As a consequence, the persistence of protective concentrations of immunoglobulin represents one of the main determining factors to achieve the herd immunity during periodic outbreaks. Neutralizing immunoglobulin (both IgM and IgG) levels rise within days to week after symptoms onset in the majority of individuals infected by SARS-CoV-2 [4]. At the very beginning of the pandemic, it has been postulated that the primary exposure to SARS-CoV-2 induces a protection towards reinfection, at least in macaques re-challenged with SARS-COV-2 after 28 days from primary exposure [5,6]. However, further data demonstrated that, unlike many other respiratory viruses resulting in immunoglobulin concentrations that last for several months or years, neutralizing immunoglobulins against SARS-COV-2 persist for about 40 days [6]. As a consequence, the risk of reinfection by SARS-CoV-2 is a well described possibility, and has been widely demonstrated in the clinical practice. After sporadically described case reports of reinfection [7-10], a definitive paper on this argument was published by To et al. [11]. The authors described a case of COVID-19 re-infection by a phylogenetically distinct strain; this finding leaded to several assumptions on long-term immunity, sampling technique standardization, viral mutation and efficacy of herd immunity. As a consequence, SARS-CoV-2 may continue to circulate among the global population despite herd immunity due to natural infection or vaccination. In particular, this case has a time gap of over 4 months, a laboratory proven different genotype resembling the European virus and the patient was also returning from Europe; all these considerations make it more likely that it is a re-infection rather than a recurrence.

Another specific issue is that, despite the mass vaccination, the presence of more than 80 genotypical variants of the virus makes at least plausible that cases of reinfection will continue to be documented. Immunoglobulin levels may not correlate with persistent viral shedding and risk of transmissibility of SARS-CoV-2 [12]. Further, the short duration of seropositivity for neutralizing antibodies raise the concern that vaccination may not result in an effective and long-term immunity against SARS-CoV-2. As a consequence, the short duration of immunity against the virus may not result in full and lasting coverage of the entire population still susceptible to COVID-19 infection. Therefore, herd immunity may not be achieved as reinfection may occur even in the presence of neutralizing antibodies.

All these considerations raise concerns that eliminating definitively the SARS-CoV-2 pandemic may not be as feasible as once supposed and, above all, that we must rely more on the prevention of the transmission to contain as much as possible the spread of infection.

References

1. Hanaei S, Rezaei N (2020) COVID-19: developing from an outbreak to a pandemic. *Arch Med Res* 51: 582-584. [Crossref]

Correspondence to: Marco Bongiovanni, Pneumology Unit, Department of Medicine, Ospedale Salvini, Garbagnate Milanese, ASST Rhodense, Milan, Italy, E-mail: mbongiovanni@asst-rhodense.it

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2. Backer JA, Klinkenberg D, Wallinga J (2020) Incubation period of 2019 novel coronavirus (2019 n-CoV) infections among travelers from Wuhan, China. *Euro surveill* p. 25. [Crossref]

3. Randolph HE, Barriero LB (2020) Herd immunity: understanding COVID-19. *Immunity* 52: 737-741. [Crossref]

4. Lofti M, Rezaei N (2020) SARS-CoV-2: a comprehensive review from pathogenicity of the virus to clinical consequences. *J Med Vir* 92: 1864-1874. [Crossref]

5. Deng W, Bao L, Liu J, Xiao C, Liu J, et al. (2020) Primary exposure to SARS-CoV-2 protects against reinfection in rhesus macaques. *Science* 369: 818-823. [Crossref]

6. Kirkcaldy RD, King BA, Brooks JT (2020) COVID-19 and postinfection immunity:limited evidence, many remaining questions. *JAMA* 323: 2245-2246. [Crossref]

7. Bongiovanni M, Basile F (2020) Re-infection by COVID-19: a real threat for the future management of pandemia? *Infect Dis* 52: 581-582. [Crossref]

8. Yuan J, Kou S, Liang Y, Zeng J, Pan Y, et al. (2020) PCR assays turned positive in 25 discharged COVID-19 patients. *Clin Infect Dis* p. 8. [Crossref]

9. Wu J, Liu X, Liu J, Liao H, Long S, et al. (2020) Coronavirus Disease 2019 test results after clinical recovery and hospital discharge among patients in China. *JAMA Netw Open* p. 3. [Crossref]

10. Ye G, Pan Z, Pan Y, Deng Q, Chen L, et al. (2020) Clinical characteristics of severe acute respiratory syndrome coronavirus 2 reactivation. *J Infect* 80: e14-17. [Crossref]

11. To KK, Hung IF, Ip JD, Chu AW, Chan WM, et al. (2020) COVID-19 re-infection by a phylogenetically distinct SARS-coronavirus-2 strain confirmed by whole genome sequencing. *Clin Infect Dis* 25: ciaa1275. [Crossref]

12. Roy S (2020) COVID-19 reinfection: myth or truth? *SN Compr Clin Med* 2: 710-713. [Crossref]