 doi:10.20944/preprints202004.0139.v1

Potential Super Spreading's of SARS-2 Pandemic. A Short Review

Anthony M. Kyriakopoulos

Nasco AD Biotechnology Laboratory, 11 Sachtouri Str, 18536 Piraeus, Greece.

Abstract

Corona viruses cause extensive SARS epidemics via super spread events (SSE). Due to variation in infection risk and heterogeneity of reproduction numbers specific distinction between SSE's and typical case events is essential. SARS transmissions unveil a complex scenario in which SSE's are shaped by multiple factors. Specific screening strategies for infection emergence within potential super spreading groups will help to efficiently control the SARS-2 pandemic and alleviate the partially effective general restriction measures.

Key words: SARS epidemiology; super spread events; efficient diagnosis to contain magnitude of SARS-2 outbreaks

Preprints (www.preprints.org) | NOT PEER-REVIEWED | Posted: 9 April 2020 doi:10.20944/preprints202004.0139.v1

1. Introduction

In many ways to control better the SARS-2 pandemic, post epidemic investigations of previous SARS should be looked in depth. SARS-2-Covid-19 is also an animal originating coronavirus that crossed species barriers to infect human (1). The initially investigated l strains of SARS-2 (covid-19) had also low potential for transmissibility and thus infectivity like in SARS-1 (1-2). SARS-1 coronavirus caused potentiated epidemics due to super spread events that elevated unexpectedly the basic and effective reproduction numbers (3). The lesson that must have been learned with SARS-1 epidemic is that even taking all restrictive contact measures to minimize below one the self sustaining threshold, these alone cannot withhold the epidemic (3).

2. The previous SARS epidemics and the SARS-2 pandemic

In many ways the reservoirs of infection of SARS-1 may be similar to SARS-2 (1-3). Identification of animal and insect vectors that transmit the disease, identification and control of alternative routes of transmission like fecal-oral route, and identification of super spreader groups of potential patients will help to minimize the epidemiology extend seen with SARS-2 worldwide. Even when trying to lower the reproduction numbers, the minimization of time from community diagnosis of infection to prompt hospital isolation was the key to control progression of epidemic of SARS-1. This was because it was important to minimize the probability that another super spread event (SSE) might occur (3).

3. The 20/80 rule

In nature, epidemics follow the 20 / 80 rule (4). Likewise in human population, due to heterogeneity of exposure to infection, the 20 % core population for reasons such as reduced immune status (e.g. haemodialysis patients) and exposure to vectors transmitting the disease (e.g. cockroaches and roof rats) (3-5) as seen with SARS-1 may contribute at least to 80% of the total transmission potential (4). What has been learned from SARS-1 is that control programs that failed to identify and provide a targeted infection diagnosis in potential SSE groups of population were inefficient to control the epidemic in population (3-4).

Preprints (www.preprints.org) | NOT PEER-REVIEWED | Posted: 9 April 2020 doi:10.20944/preprints202004.0139.v1

4. The prevention of SSE's

By preventing SSE's may enable to overcome initial low SARS virus infectiveness capability that dismantles the epidemiology attempts to statistically lower the reproduction numbers only by perusing high risk isolation and obsolete restriction of contact measures as carried out in countries like Italy and Greece (3). As seen in Beijing and Singapore the vast majority of infected individuals were barely infective and as traced for a much smaller percentage of high infectivity, only the 6 % of population was highly infectious contrasting many published SARS models (2-3). Other ways of potentiating infectivity for coronaviruses within hosts may provide explanations of enormous outbreaks (6). Cross species barrier transmission and genetic adaptation within hosts may promote infectivity of coronaviruses in humans (7), Thus focus on identification of specific potential super spreader groups within population by targeted diagnosis is urgently needed (table 1). Otherwise equivocal results like persistence of epidemic involving innocent victims isolated at home or hospital may procure (2,-4, 8-9, 11-12). In many ways, a normal infection must be distinguished from a super spread infection and different equations govern each case (3). In SARS-1 epidemic, the coronavirus infectiousness occurred mostly at late stages of infection (3-4), whereas in SARS-2, viruses are transmitted at pre-symptomatic stages (15). Likewise with H1N1 transmission (16), accurate diagnosis of covid-19 in potentially asymptomatic super spreaders within potential groups may help to contain the magnitude of large outbreaks (17).

SARS-2 coronavirus has evolved to cause a prolonged pandemic through complex epidemiology routes. In many ways this serious SARS-2 pandemic that causes both social and economic catastrophe globally, must remind us Dr John Snow, a noble obstetrician in 1854 that stopped cholera epidemic only with his high skills in preventative epidemiology and negligible technological aid. In nowadays with such high technology utilized for diagnosis, clinical observation is also essential.

5. Conclusions

SARS-2 pandemic needs containment of super spread events in a more fastidious way. This can be achieved by early diagnosis of pre-symptomatic infected individuals within potential super spreading groups. These groups however should be allocated promptly. Prevention of SARS large outbreaks is more essential, especially due to the

 doi:10.20944/preprints202004.0139.v1

unfortunate pandemic of SARS-2. The lack of efficient vaccination and therapeutic protocols makes the need of efficient prevention even more needful as SARS-2 virus follows complex infectious patterns.

Conflicts of interests

The author declares that has no competing interests.

Funding

No funding or grant was provided for this study.

Acknowledgements

I thank my family for providing moral assistance to the write up of this manuscript.

References

1. Dong N, Yang X, Ye L, Chen K, Chan EWC, Yang M, Chen S. Genomic and protein structure modelling analysis depicts the origin and infectivity of 2019-nCoV, a new coronavirus which caused a pneumonia outbreak in Wuhan, China. (2020). https://www.biorxiv.org/content/10.1101/2020.01.20.913368v1#disqus_thread doi: https://doi.org/10.1101/2020.01.20.913368
2. Lloyd-Smith JO, Schreiber SJ, Kopp PE, Getz WM. Superspreading and the effect of individual variation on disease emergence. Nature. 2005 Nov 17;438(7066):355-9. doi: 10.1038/nature04153
3. Riley S, Fraser C, Donnelly CA, Ghani AC, Abu-Raddad LJ, Hedley AJ, Leung GM, Ho LM, Lam TH, Thach TQ, Chau P, Chan KP, Lo SV, Leung PY, Tsang T, Ho W, Lee KH, Lau EM, Ferguson NM, Anderson RM. Transmission dynamics of the etiological agent of SARS in Hong Kong: impact of public health interventions. Science. 2003 Jun 20;300(5627):1961-6. doi:10.1126/science.1086478
4. Woolhouse MEJ, , Dye C, Etard J_F, , Smith T, et al. Heterogeneities in the transmission of infectious agents: Implications for the design of control programs. PNAS January 7, 1997 94 (1) 338-342; https://doi.org/10.1073/pnas.94.1.338
5. Ng SK. Possible role of an animal vector in the SARS outbreak at Amoy Gardens. Lancet. 2003 Aug 16;362(9383):570-2. doi:10.1016/S0140-6736(03)14121-9
6. Taguchi F, Matsuyama S. Soluble receptor potentiates receptor-independent infection by murine coronavirus. J Virol. 2002;76(3):950–958. doi:10.1128/jvi.76.3.950-958.2002
7. Perlman S, Netland J. Coronaviruses post-SARS: update on replication and pathogenesis. Nat Rev Microbiol. 2009;7(6):439–450. doi:10.1038/nrmicro2147
8. Shen, Z., Ning, F., Zhou, W., He, X., Lin, C., Chin, D. P., Zhu, Z., & Schuchat, A. (2004). Superspreading SARS events, Beijing, 2003. Emerging infectious diseases, 10(2), 256–260. https://doi.org/10.3201/eid1002.030732
9. Outbreak of Severe Acute Respiratory Syndrome (SARS) at Amoy Gardens, Kowloon Bay, Hong Kong Main Findings of the Investigation. https://www.info.gov.hk/info/sars/pdf/amoy_e.pdf

10. Liu, W., Fontanet, A., Zhang, P. H., Zhan, L., Xin, Z. T., Tang, F., Baril, L., & Cao, W. C. (2006). Pulmonary tuberculosis and SARS, China. Emerging infectious diseases, 12(4), 707–709. https://doi.org/10.3201/eid1204.050264
11. Kwan BC, Leung CB, Szeto CC, Wong VW, Cheng YL, Yu AW, Li PK. Severe acute respiratory syndrome in dialysis patients. J Am Soc Nephrol. 2004 Jul;15(7):1883-8.
12. Yap FH, Gomersall CD, Fung KS, Ho PL, Ho OM, Lam PK, Lam DT, Lyon DJ, Joynt GM. Increase in methicillin-resistant Staphylococcus aureus acquisition rate and change in pathogen pattern associated with an outbreak of severe acute respiratory syndrome. Clin Infect Dis. 2004 Aug 15;39(4):511-6.
13. Stein RA. Super-spreaders in infectious diseases. Int J Infect Dis. 2011 Aug;15(8):e510-3. doi: 10.1016/j.ijid.2010.06.020.
14. Perlman, S., & Netland, J. (2009). Coronaviruses post-SARS: update on replication and pathogenesis. Nature reviews. Microbiology, 7(6), 439–450. https://doi.org/10.1038/nrmicro2147
15. Wei WE, Li Z, Chiew CJ, Yong SE, Toh MP, Lee VJ. Presymptomatic Transmission of SARS-CoV-2 — Singapore, January 23–March 16, 2020. MMWR Morb Mortal Wkly Rep. ePub: 1 April 2020. doi: http://dx.doi.org/10.15585/mmwr.mm6914e1external icon.
16. Gu, Y., Komiya, N., Kamiya, H., Yasui, Y., Taniguchi, K., & Okabe, N. (2011). Pandemic (H1N1) 2009 transmission during presymptomatic phase, Japan. Emerging infectious diseases, 17(9), 1737–1739. https://doi.org/10.3201/eid1709.101411
17. Thompson, R. N., Gilligan, C. A., & Cunniffe, N. J. (2016). Detecting Presymptomatic Infection Is Necessary to Forecast Major Epidemics in the Earliest Stages of Infectious Disease Outbreaks. PLoS computational biology, 12(4), e1004836. https://doi.org/10.1371/journal.pcbi.1004836

Preprints (www.preprints.org) | NOT PEER-REVIEWED | Posted: 9 April 2020 doi:10.20944/preprints202004.0139.v1

Table 1.

Potential groups of coronaviruses super spreaders within human population*

Population Group	Potential route of transmission
Hepatitis vurus positive patients	Airborne (6)
Pulmonary tuberculosis positive patients	Airborne (10)
HIV positive patients	Airborne and urine - fecal oral (13)
Patients receiving haemodialysis	Airborne (droplets by nebulizer) and fecal oral (2,3,11)
MRSA *Staphylococcus aureus* acquisition	Constant Worn Glove Contact Transmission (12)
Rhinovirus co-infections	Airborne (13)
Gastrointestinal *(Salmonella enteritis)* co-infections	Fecal – oral transmission (9,13)
Frequent contact with wild animal reservoirs (including domestic animals) and birds**	Airborne and fecal – oral (14)
Construction area workers	Air particles (2-3, 13)

*In both community and hospital environment. **Including slaughter houses, pet shops, animal and bird collectors and breeders, cow and pig farmers.