

# What was said about COVID-19 and what we heard and what we saw!

Dear Editor,

We reviewed the literature, searching about the followings and compare them to our local clinical findings, came to few local differences.

1. What has been said and done, about the means of transmission:

In the early days of pandemic, nearly everything was accused as presumptive transmission objects, and disinfection protocols on a large scale were designed and use of too many detergent materials got so extended that, they could be themselves a health hazard.<sup>[1]</sup>

Humanity may be in fight and involve for a long time with coronavirus and others. Exaggerated and unnecessary prophylactic programs may cause the waste of health system deposits and staff energy. We have to see the most important ways of transmission and make prophylactic measures easier and much more practical. Now believing that only human breathing and close proximity are the main modes of transmission.

2. Regarding the high-risk groups, pregnancy<sup>[2]</sup>, cardiac diseases, immunosuppression, obesity, and chronic kidney diseases, long-standing diabetes mellitus<sup>[3]</sup>, Chronic obstructing pulmonary disease and hypertension along with age over 60 years have been accepted as high risk medical conditions.<sup>[4]</sup>

What we saw in addition to the above, but still unanswered:

We encountered young patients who did not have any of the defined risk factors, but became severely ill and died. We think that, they are the unluckiest group, who do not have any of the above defined risk factors but may have other possibilities for catching intense disease:

- Perhaps, the immune system of this group, never have come in touch with any of the strains of the corona group. Hence, there is no known antigen fragments to be presented to immune system in the initial encounter
- Or these patients received a high dose of the virus in the first encounter, supposed up to now these people, mostly are among health care givers
- May it be genetic predisposition or possibility of diversity in the virus subtype? All the above options

can be equally important and can be subject of discussion.

3. It was said and written about, the serial events going ahead to cause death:

Mortality and the crisis period of the disease have been attributed to cytokine storm syndrome (CSS).

Although in the inflammatory phase of disease, CSS is a prominent cause of death, but we should see another significant pathology. Based on the imaging, pulmonary lesions are found to be in the peripheral areas of the lung and mostly in the right lung, and in the early stages, we have no central lesions. Thus, the most likely hypothesis it is bronchogenic dispersion, with the virus first encountering the immune system at the alveolar level. In this case, even if all the alveoli are involved and the lung is completely white, the direct cause of death will be respiratory failure and ARDS.

CSS also may be a terminal event of sepsis with bacterial agents. We think, prolonged hospital stay makes older groups or people with an underlying disease,<sup>[5]</sup> much more prone to sepsis and death while they have come primarily with corona infection.

Discharging patients, who have passed the inflammatory phase and are in a well condition, and transfer them to home, may decrease the risk of nosocomial infections and sepsis.

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

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**Submitted:** 09-Aug-2020; **Revised:** 08-Sep-2020;


**Accepted:** 31-Jan-2021; **Published:** 30-Sep-2021

## REFERENCES

1. Zou L, Ruan F, Huang M, Liang L, Huang H, Hong Z, *et al.* SARS-CoV-2 viral load in upper respiratory specimens of infected patients. *N Engl J Med* 2020;382:1177-9.

2. Liu H, Wang LL, Zhao SJ, Kwak-Kim J, Mor G, Liao AH. Why are pregnant women susceptible to COVID-19? An immunological viewpoint. *J Reprod Immunol* 2020;139:103122.
3. Pal R, Bhansali A. COVID-19, diabetes mellitus and ACE2: The conundrum. *Diabetes Res Clin Pract* 2020;162:108132.
4. Henry BM, Lippi G. Chronic kidney disease is associated with severe coronavirus disease 2019 (COVID-19) infection. *Int Urol Nephrol* 2020;52:1193-4.
5. Canna SW, Behrens EM. Making sense of the cytokine storm: A conceptual framework for understanding, diagnosing, and treating hemophagocytic syndromes. *Pediatr Clin North Am* 2012;59:329-44.

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Quick Response Code:	Website: <a href="http://www.jmsjournal.net">www.jmsjournal.net</a>
	DOI: 10.4103/jrms.JRMS_948_20

**How to cite this article:** Rostami M, Shirani K, Haghighipour S, Yazdani MR. What was said about COVID-19 and what we heard and what we saw! *J Res Med Sci* 2021;26:84.

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