In COVID-19, do we need surfactant therapy?

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Abstract :

COVID-19 started from Wuhan-china by end of 2019 and then spread all over the world. WHO declared it as a pandemic.

A lot of theories about its pathogenesis were published suggesting its ability via spike protein S to bind to ACE 2 in type 11 pneumocytes as lock and key model then virus genome transferred within cell to begin replication and millions of copy are produced and start to infect other cells, There is associated inflammatory response start by macrophages to engulf this foreign antigen and chemotactic and cytokines production followed by increase capillary permeability up to cytokine storm and death.(1) Protocols of treatment:

Varying from mild cases with just supportive treatment with self-isolation to moderate, severe and critically ill cases which becomes ventilated and little percentage will survive of ventilated cases.(6)

Rational:

In premature neonates with a primary surfactant deficiency , there is ARDS and surfactants is the main line of therapy. Now it is well known that the virus is attached to and is replicated within type 11 pneumocytes, the main function of these cells is production of surfactants which decrease surface tension and keep the alveoli patent without collapse. In this condition there is a secondary surfactant deficiency which may play a role in pathogenesis and deterioration of patient clinical condition.

So, I suggest that treatment by surfactant therapy either derived from endogenous or exogenous source in severe cases can improve clinical condition, delay or avoid patient ventilation by refilling debilitated surfactants and lavage of bronchial tree, this directly decrease mortality rate.

Keywords : covid_19; surfactant therapy; ARDS

Introduction:

COVID-19 started from Wuhan-china by end of 2019 and then spread all over the world. WHO declared it as a pandemic.

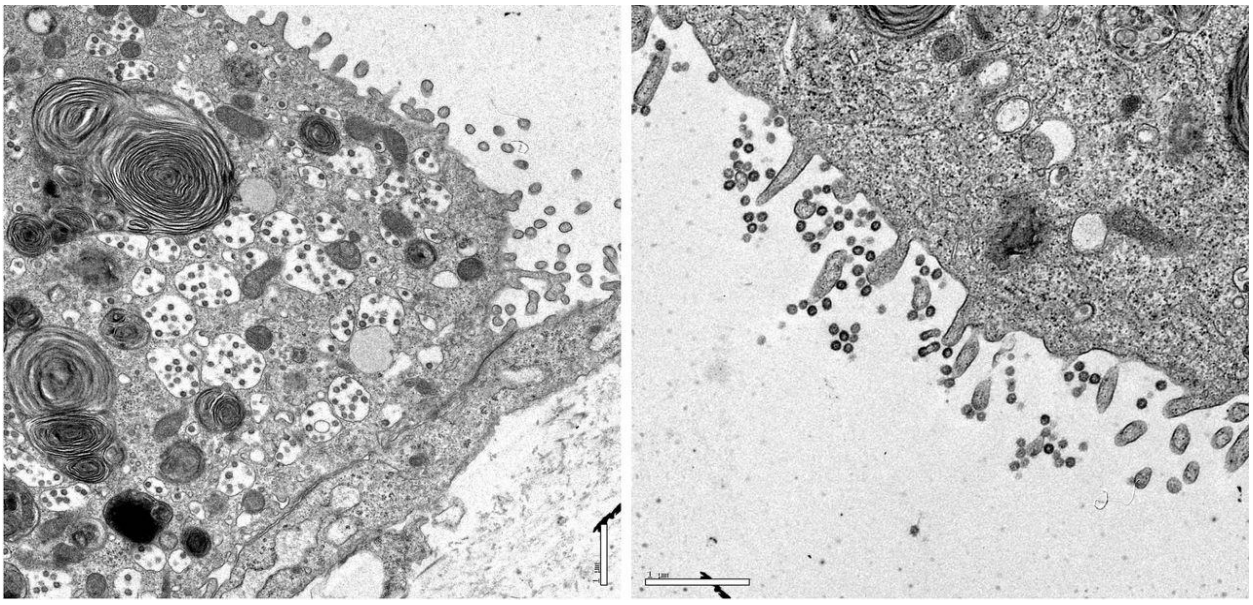
A lot of theories about its pathogenesis were published suggesting its ability via spike protein S to bind to ACE 2 in type 11 pneumocytes as lock and key model then virus genome transferred within cell to begin replication and millions of copy are produced and start to infect other cells, There is associated inflammatory response start by macrophages to engulf this foreign antigen and chemotactic and cytokines production followed by increase capillary permeability up to cytokine storm and death.(1)

Also hypercoagubale state was observed and vascular pulmonary thrombosis as found in many patients. Recently, the ability of the virus to attack β chain of hemoglobin and dissociation into myoglobin and

porphyrin leading to inability to carry oxygen and persistent hypoxia occur with precipitation of heam molecule within lung tissue with initiation of more tissue damage and hypoxia giving the CT findings .(2)

Unfortunately, about 20% of the infected patients will progress to severe form of disease and will develop pulmonary infiltrates . Initial estimates of the fatality rate are around 2%, but this varies markedly with age.(3)

In this severe form of disease the virus reaches the gas exchange units of the lung and infects alveolar type II cells. Both SARS-CoV and influenza preferentially infect type II cells compared to type I cells (4)



Figure(1):Human alveolar type 11cells infected with SARS_COV2

Human alveolar type II cells infected with SARS-CoV. Human type II cells were isolated, cultured in vitro, and then infected with SARS-CoV. Viral particles are seen in double membrane vesicles in the type II cells (left panel) and along the apical microvilli (right panel)(5)

Protocols of treatment:

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So, I suggest that treatment by surfactant therapy either derived from endogenous or exogenous source in severe cases can improve clinical condition, delay or avoid patient ventilation by refilling debilitated surfactants and lavage of bronchial tree, this directly decrease mortality rate.

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