

Pathogenesis and Mechanisms of Covid Transmission 19.

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Abstract

March 11, 2020. The World Health Organization has declared Coronavirus Disease 2019 (COVID-19) a pandemic. Despite a large number of scientific publications concerning the clinical picture and treatment methods, there is insufficient data on the structural changes of internal organs in COVID-19. Coronavirus infection 2019 (covid-19) caused by SARS-CoV-2 follows a two-phase disease pattern, which probably results from a combination of early and late symptoms: a viral response phase and a second inflammatory phase [3]. COVID-19 infection is caused by Severe acute respiratory syndrome-related coronavirus 2 (SARS-CoV-2), which is tropic to angiotensin-converting enzyme receptor 2 (ACE2). Interaction with this receptor is the initial link in pathogenesis. Morphological pattern is similar to pneumonias caused by SARS-CoV and Middle East respiratory syndrome coronavirus (MERS-CoV): at initial stage lung shock pattern develops, later it ends with fibrosis and organizing pneumonia. One of the most severe complications is acute respiratory distress syndrome (ARDS).

Keywords:

new coronavirus infection, pregnant women, newborns, pneumonia, Covid 19.

Introduction: Severely ill or immunocompromised patients may have relatively prolonged viral release, and some patients may have intermittent RNA release. A combination of preventive measures, such as physical distancing and testing, tracking, and self-isolation, is still necessary [1,6]. Pathogenesis. Virus entry and interaction with target cells SARS-CoV-2 binds to ACE 2, the target cell receptor. Active replication and release of the virus in lung cells leads to nonspecific symptoms such as fever, myalgia, headache, and respiratory symptoms. In an experimental hamster model, the virus causes temporary damage to olfactory epithelial cells, leading to olfactory dysfunction, which may explain the temporary loss of taste and smell commonly seen in covid-19. The distribution of ACE 2 receptors in

various tissues may explain the foci of infection and patient symptoms. For example, the ACE 2 receptor is found in the epithelium of other organs, such as the intestine and endothelial cells of the kidneys and blood vessels, which may explain gastrointestinal symptoms and cardiovascular complications. Lymphocytic endotheliitis has been detected in pathological examination of the lungs, heart, kidneys, and liver, as well as in liver cell necrosis and myocardial infarction in patients who have died of COVID-19. These data show that the virus directly affects many organs, as has been seen with SARS-CoV-1 and influenza.

Much remains unclear. Are abnormal airway changes or endothelial dysfunction the result of direct viral infection, cytokine dysregulation, coagulopathy, or are they multifactorial? And does direct viral invasion or coagulopathy directly contribute to some ischemic complications such as ischemic infarcts? This and more will require further research to elucidate.

Immune response and disease spectrum. After virus entry, an initial inflammatory response attracts virus-specific T cells to the site of infection, where infected cells are destroyed before the virus spreads, resulting in recovery in most people. In patients with severe disease, SARS-CoV-2 causes an abnormal host immune response [3, 4]

Infection resulting from direct or indirect contact with the nasal, conjunctival, or oral mucosa. The target host receptors are found mainly in the human respiratory epithelium, including the oropharynx and upper respiratory tract. The conjunctiva and gastrointestinal tract are also susceptible and can serve as portals of transmission.

The risk of transmission depends on factors such as the nature of contact, the environment, the infectious burden of the host, and socioeconomic factors [5]. Most transmission occurs through close contact (15 minutes face-to-face and within 2 m), and transmission is particularly effective within households or in organized groups [7]. The frequency of secondary attacks in the home (the proportion of persons who become infected in a susceptible contact group with a primary case) ranges from 4% to 35%. An infected person increases the risk of infection, but isolating an infected person from the family results in a lower risk of infection. Other high-risk activities include eating in close proximity to an infected person, sharing food, and participating in group activities. The risk of infection increases significantly in indoor compared to outdoor conditions. Aerosol transmission may still be affected by prolonged stay in crowded, poorly ventilated rooms (this means that transmission can occur at a distance > 2 m) [7].

Some patients have a high risk for septic shock and multiple organ dysfunction. For example, the cardiovascular system is often involved in early COVID-19 disease, which is reflected in the release of highly sensitive troponin and natriuretic peptides. In the clinical context, coagulopathies, also focal intra-alveolar bleeding and/or the presence of platelet-fibrin thrombi in small arterial vessels play an important role. Cytokines usually mediate and regulate immunity, inflammation and hematopoiesis; however, further exacerbation of the immune response and accumulation of cytokines in other organs in some patients can cause extensive tissue damage, or cytokine release syndrome (cytokine storm), leading to capillary rupture, clot formation and organ dysfunction.

Mechanisms underlying different clinical disease outcomes. Clinical outcomes are influenced by risk factors (premorbid background) such as older age, male gender, and comorbidities, as well as virus-related factors (e.g., viral load kinetics), host immune response, and potential cross-immune memory from previous exposure to seasonal coronaviruses. Gender-related differences in immune response have

been reported, showing that males initially had more cytokines and chemokines of innate immunity in plasma than females. In contrast, women had markedly stronger T-cell activation than men, and T-cell activation decreased with age among diseased men, which was consistent among female patients. These findings suggest that the adaptive immune response may be important in determining clinical outcome, as older age and male gender are associated with an increased risk of severe disease and mortality. Elevated levels of proinflammatory cytokines correlate with the incidence of severe pneumonia and increased pulmonary opacity ("frosted glass" effect in the lungs) on radiography. Identified pathomorphological features of the inflammatory process in COVID-19 (priority of endothelial damage with micro- and macrothrombosis, relatively late development of the exudative phase of inflammation and propensity to develop pneumofibrosis) predetermine greater duration of therapy and the need for respiratory rehabilitation, mainly aimed at pulmonary recruitment. At the same time, the results obtained raise a number of relevant questions about the feasibility and duration of the use of a number of drugs.

Literature.

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