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Research Article

PATHOGENESIS OF COVID-19 AND THE BODY'S RESPONSES

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ABSTRACT

Children are infected with the virus without suffering a serious disease and represent an important source of infection. It has been experimentally proven that young mice respond to lung tissue damage from viral infection through prostaglandins, while adult mice succumb. The angiotensin-converting enzyme (ACE) 2 receptor is particularly abundant on the cells of the lower lung pathways, whose situation explains the high incidence of bronchitis and pneumonia related to the severe infection of COVID-19. A fall in ACE2 activity in the elderly is partly responsible for the decreased ability to reduce the inflammatory response with old age. The reduction of ACE2 receptors in older adults puts them in a position where they are unable to cope with COVID-19. In Italy from the details of the medical records of the current hospitalized as well as those discharged healed and unfortunately the victims do not seem to have any foreigner in the sense of a non-EU citizen. Non-EU citizens are all covered by a tuberculosis vaccine which is part of a coverage protocol provided by the Local Health Unit. It seems that flu vaccination favors coronavirus infection, even greater than 36% as reported by an American military study. Both meningococcal and pneumococcal disease have been associated with the activity of influenza and respiratory syncytial viruses. The Istituto Superiore della Sanità recently stated that few deaths are from coronavirus and instead most put of them from other pathologies (cardiovascular, cancer, diabetes, etc.). This suggests that the overall clinical consequences of COVID-19 could ultimately be similar to that of severe seasonal flu, which has a fatality rate of approximately 0.1%, or pandemic influenza such as that of 1957 or 1968, rather than those of SARS or MERS, characterized respectively by a fatality of 10% and 36%.

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INTRODUCTION

According to the experience of the first SARS and of the MERS, the children were not exposed to the civet cat and camels in a similar way (1). It was thought that the same fact could take place with the SARS from COVID-19 (2). Indeed children are infected with the virus without suffering a serious disease and represent an important source of infection. The virus is found in their rectal swabs.

Growing with age many specific cells of the immune system are no longer active and therefore the body loses its ability to respond effectively. In fact, it has been experimentally proven that young mice respond to lung tissue damage from viral infection through prostaglandins, while adult mice succumb. The juvenile immune system and its efficient T Helper cells respond to SARS COVID 2. The Helper cell's CD4 lymphocytes stimulate B cells to produce antibodies against the virus and control infection. In this case Th2 lymphocytes are able to control the inflammatory response caused by the viral infection, preventing an exuberant

and delayed reaction as occurs in adults. The different hormonal structure and the same prostaglandins favor the female subject against the coronavirus responsible for the current pandemic.

Another important discussion concerns the ACE2 receptor, that is, the angiotensin-converting enzyme 2. Both the first SARS and the current SARS have the same cellular entry route through this receptor for coronaviruses (3). The receptor is particularly abundant on the cells of the lower lung pathways, whose situation explains the high incidence of bronchitis and pneumonia related to the severe infection of COVID-19(4). The same receptor is abundantly represented on the mouth and tongue facilitating the viral entry of the host organism. Despite its reduction with adulthood, the ACE2 enzyme is an important regulator of the immune response, in particular inflammation protects mice against acute lung damage triggered by sepsis. In 2014 it was shown that the ACE2 enzyme protects against lethal avian influenza. Some of the best-performing patients had high levels of the protein in their serum. By blocking the gene for ACE2, severe lung

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damage was observed in mice infected with H5N1, while with the treatment of mice with human ACE2, lung damage decreased.

A fall in ACE2 activity in the elderly is partly responsible for the decreased ability to reduce the inflammatory response with old age. The reduction of ACE2 receptors in older adults puts them in a position where they are unable to cope with COVID-19.

Primary complex and tuberculosis vaccine

In Italy from the details of the medical records of the current hospitalized as well as those discharged healed and unfortunately the victims do not seem to have any foreigner in the sense of a non-EU citizen (5). It seems that these subjects, which for some municipalities in the North are even the majority, may have a normal flu-like syndrome (from coronavirus) without developing any criticality. It seems that they behave like Italian children who did not get pneumonia because they were vaccinated against tuberculosis, a vaccination that lasts for twenty years. After twenty years they begin to get sick with tuberculosis as now with COVID-19. Non-EU citizens are all covered by a tuberculosis vaccine which is part of a coverage protocol provided by the Local Health Unit.

Viruses have no prejudices neither of sex, nor of census, nor of ethnicity. About 90% of people infected with Mycobacterium tuberculosis have an asymptomatic TB infection (also called LTBCI, from latent tuberculosis infection), and only 10% chance in life that a latent infection develops in TB (6).

Tuberculous infection begins when mycobacteria reach the lung alveoli, where they attack and replicate within the alveolar macrophages. The primary site of infection in the lungs is called the Ghon outbreak. The bacteria are collected by the dendritic cells, which do not allow their replication but which can transport the bacilli to the local mediastinal lymph nodes. The primary lesion of the mycobacterium accompanied by satellite adenopathy represents the "primary complex", in which the bacilli remain walled up without giving rise to clinical manifestations, but can resume their pathological activity and spread in the organism especially following an immunodeficiency of the individual. During the World Wars it was the colored troops who were cut down by the White Tuberculosis and not vice versa. Obviously it could also be that on return a defed white man, without adequate food, stressed for the war, could in turn contract it from foreigners but the norm was that the "colored" soldiers contracted it from the Whites (6).

In the Sierra de Ecuador, normally everyone received TB vaccination, only in recent years there has been discussion about whether to make it optional. This would confirm the observation that in the Sierra cases of manifest infection of COVID-19 are very few.

In Australia tests on 4 thousand doctors and nurses with the tuberculosis vaccine took place www1.racgp.org.au (The Royal Australian College of General Practitioners).

Viral interference

In Italy there are multiple factors that may have interacted together and that explain the situation. Contacts with the Chinese virus are presumed to have been greater in the Center-North than in the Center-South. Add to this the concomitance of the environmental and climatological situations, different between North and South of Italy, even going as far as to hypothesize that over the weeks an indigenous Padan coronavirus has formed, different from the Chinese one. Other possibilities emerge from the situations of Bergamo and Brescia above all, where it is assumed that the circulation of other viruses may have facilitated the action of SARS-Cov-2. The problem, however, was above all upstream: namely, not having enough intensive care beds, mostly occupied already because of the annual flu. It seems that flu vaccination favors coronavirus infection, even greater than 36% as reported by an American military study: (7). On the other hand, since there has been a recent emerging meningitis, 34,000 people have been vaccinated between Brescia and Bergamo. There has been a publication of Dutch scholars printed by a scientific journal of the University of Cambridge in which both meningococcal and pneumococcal disease have been associated with the activity of influenza and respiratory syncytial viruses (8).

COVID-19 mortality rate

The Istituto Superiore della Sanità recently stated that few deaths are related to coronavirus and instead most of them to other pathologies (cardiovascular, cancer, diabetes, etc.), 29 out of 909.

The mortality rate associated with COVID-19 may be considerably less than 1%, rather than the 2% reported by some groups, as stated by Anthony Fauci of the U.S. National Institute of Allergy and Infectious Diseases based on a report focusing on 1099 patients with laboratory confirmed COVID-19 from 552 Chinese hospitals(9). These patients had a broad spectrum of disease severity, and if it is assumed that the number of asymptomatic or minimally symptomatic cases is several units of magnitude greater than that of the reported cases, the fatality rate of the disease would fall far below 1%(9).

This suggests that the overall clinical consequences of COVID-19 could ultimately be similar to that of severe seasonal flu, which has a fatality rate of approximately 0.1%, or pandemic influenza such as that of 1957 or 1968, rather than those of SARS or MERS, characterized respectively by a fatality of 10% and 36%. Passive immunotherapy has been suggested for coronavirus (10).

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