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Editorial

SARS-CoV-2 Epidemic and Diabetes

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An editorial related to the recent SARS-CoV-2 epidemics in a diabetes/metabolism journal might seem strange. However, the implications for diabetes subjects, usually defined as a vulnerable population in this respect [1], make worth this endeavor. Moreover, the SARS-CoV-2 epidemic comes on top of the long-time recognized "diabetes epidemic" that does not show signs of relenting. Thus, the latest International Diabetes Federation (IDF) data show that 463 million people are affected worldwide, an increase of 38 million compared to 2017 [2]. In addition, total healthcare costs associated with diabetes are estimated to around 760 billion USD [2].

In December 2019, the first cases of a new form of atypical pneumonia were reported in subjects visiting/associated with the Huanan Seafood Wholesale Market commercializing live animals. The epicenter of the outbreak was located in Wuhan, Hubei region, China [3]. Very soon, Chinese researchers identified a coronavirus from pneumonia patients as the pathogen agent and designated it as 2019-nCoV, better known as SARS-CoV-2 [4], while the disease induced by this virus was called COVID-19 (Corona Virus Disease – 2019) [5]. Already at the end of January 2020, the World Health Organization (WHO) declared COVID-19 a public health emergency, an epidemic in China, and a global pandemic in March 2020 [6]. Most recent estimates (but these figures will be widely surpassed at the time of publication of this material) indicate a total of almost 156,000 cases worldwide with 5,800 fatalities and approximately 75,000 recovered cases [7].

Coronaviruses affecting humans and other vertebrates for millennia are a group of single-stranded RNA viruses belonging to the family of Coronaviridae [8]. They are continually circulating in humans and are responsible for up to 30% of respiratory viral infections, usually mild respiratory diseases [9], but they can also induce digestive or neurologic diseases [8]. They became the focus of the scientific community in 2002 due to the epidemic of Severe Acute Respiratory Syndrome (SARS) induced by another member of the family – the SARSr-CoV virus and subsequently that of the Middle-East Respiratory Syndrome (MERS) induced by the MERSr-CoV [10], viruses with which SARS-CoV-2 share at least 70% genetic similarity.

Both SARSr-CoV and MERSr-CoV were transmitted from animals to humans and caused severe pulmonary syndromes in affected individuals. The main antigenic structure on the coronavirus envelope is a spike S protein [11]. This protein strongly interacts with the human Angiotensin-Converting Enzyme 2 (ACE2) that is expressed mainly in the lower respiratory tract and on alveolar epithelial cells. The attachment of the virus to the host cells depends on the binding of the S protein to the cell receptor, while the entry of the virus in the target cell is dependent on the S protein priming by cellular proteases, specifically the serine protease TMPRSS2 [9].

Estimates regarding the incubation period range from 1 to 14 days [12], with a mean of 5 days [13], but this is not yet fully elucidated. The most typical clinical signs include fever, sore throat, cough (productive or not), myalgia, and fatigue. When present, dyspnea is a sign of gravity. Upper respiratory tract signs such as runny nose and sneezing are less typical. Digestive symptoms (including diarrhea, vomiting, and abdominal pain) were also reported in up to 10% of patients, with the possibility of virus elimination in the stools and the fecal-oral transmission route [14]. However, the primary route of human-to-human transmission is

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via respiratory droplets and direct contact with surfaces contaminated with viral particles [10].

Preliminary data from the Chinese patients show that the vast majority of cases (~80%) are mild, 14% are severe (with pneumonia, respiratory distress, and hypoxemia), and 5% are critical (respiratory failure, multiple organ failure, septic shock) [5]. Severe and critical cases might have a low fever or even no fever. There are also cases with very mild symptoms and possibly asymptomatic cases [13] that could increase the probability of disease spreading in communities.

The main laboratory findings include a low lymphocyte count and increased C Reactive Protein (CRP) levels. Severe cases associate findings suggestive for organ damage (increased liver enzymes, muscle enzymes, creatinine, troponin, D-dimers, and others) [10]. Chest imagining in patients with pneumonia indicates small patchy infiltrates and interstitial changes in the initial stages and larger infiltrative shadows in both lungs in later stages [10]. Positive diagnosis in suspected patients is made by identifying viral RNA sequences using the real-time polymerase chain reaction (RT-PCR) technique or direct gene sequencing on biological samples, including nose/throat swabs, sputum, alveolar lavage or other samples [15, 16].

Currently, there is no specific antiviral treatment for SARS-CoV-2. General recommendations include supportive measures, prompt oxygen therapy by nasal catheter/mask at first signs of oxygen desaturation, antibiotic treatment only in cases of secondary bacterial supra-infection [10]. For severe cases, specific intensive care methods (including but not limited to invasive mechanical ventilation, circulation support, glucocorticoid use) should be promptly initiated [17].

Reported mortality rates vary widely. A very recent report in Lancet Infectious Diseases indicates that up to March 1st, 2020 mortality rate in China was 3.6% while outside China, the mortality rate was 1.5% [18].

Preventive measures to prevent the human spreading of the disease are of paramount importance. Preliminary data show that SARS-CoV-2 can be inactivated by heat, chloride disinfectants, 75% ethanol, and chloroform but not by chlorhexidine [10]. General recommendations include washing hands regularly and thoroughly, avoiding touching the face (specifically the eyes, nose, and mouth) before washing/disinfecting hands, cleaning and disinfecting objects/surfaces that are touched frequently, and trying to avoid close contact with subjects with symptoms of respiratory illness.

Moving to the issue of COVID-19 risk in diabetes subjects, current estimates indicate that diabetes is the second most common condition present in infected subjects. Thus, a report from Wuhan, China, indicates that 30% of COVID-19 patients had hypertension, and 19% had diabetes [19]. In addition, diabetes was associated with a death OR of 2.85 (95% CI 1.35 to 6.05) compared to non-diabetes subjects. Another recent report, also from Wuhan, indicated that 19% of patients who developed acute respiratory distress syndrome (ARDS) had diabetes (compared to only 5% of patients without ARDS) [20].

What can we do to improve the prognostic of diabetes subjects facing the SARS-CoV-2 epidemics? The recommendations for the general population should be strongly emphasized to our diabetes population. In addition, education instructions for sick days should be reminded and reinforced. Extra attention to good glycemic control could boost immunity and resistance to viral diseases. Proper hydration and insulin dose adjustments are essential in the presence of fever and flu-like symptoms. Rules for the proper treatment of hypoglycemic episodes should also be reminded. Besides, patients should be instructed to make a proper supply of diabetes medications in the case of quarantine that could last for several weeks. For people living alone, making sure that a relative or neighbor knows the patient has diabetes and could assist in case of an emergency is, of course, important. Finally, close contact with the diabetes healthcare team should be enforced.

Conflict of Interest

The author declares no conflict of interest.

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