Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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OPINION

COVID-19 and Dysfunctional Endothelium: The Mexican Scenario

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The pandemic of a new coronavirus started in December 2019 in Wuhan China and is the cause of a high percentage of the world population infected with symptoms of the Severe Acute Respiratory Syndrome (SARS), now SARS-CoV-2. The World Health Organization (WHO) named it COVID19 disease and alerted countries with vulnerable health systems to establish an urgent response (1). The clinical conditions detected in infected patients are quite similar to those observed during previous pandemics, SARS-CoV in 2003 and MERS-CoVA in 2012. Age of people infected with COVID-19 ranges from 20–80 years old although most deaths occur in patients older than 60 yearold. Moreover, death is associated with chronic diseases such as hypertension, diabetes and vascular diseases (2). However, after the pandemic spreads in America, it has been observed, particularly in Mexico, that deaths occur in patients at a mean of 40–59 years old (3). According to the WHO, Mexico is between the world leading ranks in terms of chronic diseases and the consequent endothelial dysfunction. Is there an explanation for this apparent earlier age for death in Mexican patients? It has been reported that patients with COVID-19 may present venous thromboembolic disease or disseminated intravascular coagulopathy, events involving hemostatic elements as wells as the innate immune response (4). Endothelial dysfunction leads to platelet and leukocyte activation, abnormalities in the anticoagulant and fibrinolytic mechanisms and the resulting abnormalities of the blood coagulation tests and increased plasma D-dimer, circulating thrombin and activated protein C, closely associated to endothelial dysfunction, there is also increased inflammatory cytokines in which tumor necrosis factor α (TNF-α) has a predominant role. Patients with recurrent unprovoked venous thromboembolic disease also have both, endothelial dysfunction recognized from the early endothelial colony-forming cells (a subpopulation of circulating endothelial progenitor cells), and increased TNF-α synthesis which induces a chronic inflammatory state that may resemble the cytokine storm identified in patients with COVID-19 (5). It seems quite apparent that SARS-CoV-2 may induce endothelial dysfunction as suggested by the increased expression of the human angiotensin-converting enzyme type 2 (ACE2), a receptor necessary for the entry of the virus (6). Perhaps, deaths occurring in Mexico at younger age as compared with other countries may be related to the high frequency of vascular risk factors and the consequent endothelial dysfunction (Figure 1). Such chronic conditions may worsen as soon as the virus infects a young patient with chronic endothelial dysfunction. An old epidemic with a new face?

Conflict of Interest

We declare no competing interests.

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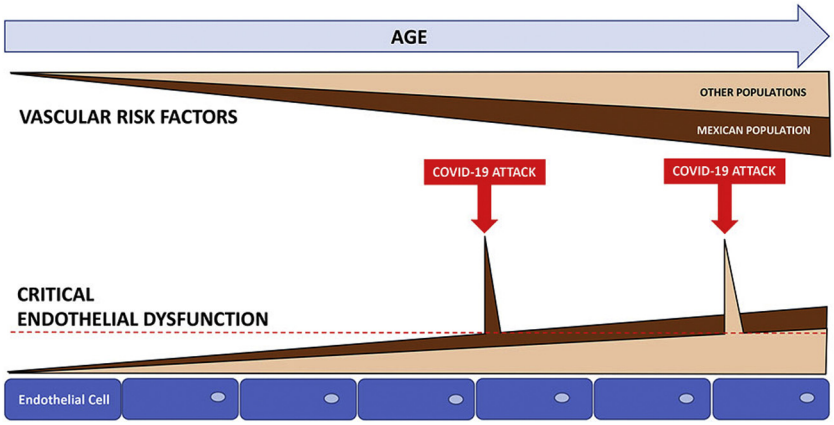

Figure 1. As the epidemics of obesity and related comorbidities (risk factors), appears earlier in the Mexican population, endothelial cell dysfunction also appears in youngest patients. When a severe trigger event affects a patient with increased vascular risk factors, namely COVID-19 infection, the critical level of endothelial cell dysfunction also appears earlier in that individual. The relationship between high frequency of vascular factors, COVID-19, and endothelial cell dysfunction may explain the deaths at a younger age in Mexico as compared with other populations.

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