Invited editorial

COVID-19 and the cardiovascular system

The COVID-19 pandemic is raging on, with impressive numbers of cases and deaths. As of 19 May 2020, there are 4,731,458 confirmed cases of COVID-19 in the world, of which 112,637 in the last 24 hours; number of deaths is 316,169 , of which 4 322 in the day before last (1) . Regions most affected are the Americas, with 2, 082,945 cases, or 44% of all cases in the world, and Europe, with 1,909,592 cases, or 40% of all reported cases. Mortality rates are, so far, 6% and 8.8%, in the Americas and Europe, respectively (1).

The new coronavirus, SARS-CoV-2, causes an acute upper respiratory tract infection in around 80% of patients; in 5 to 20% it may cause severe disease, especially involving the lungs, leading to hypoxemia and acute respiratory failure (2, 3). The disease COVID-19 manifested as a critical condition, leading to intensive care treatment, in 5% of Chinese patients, and in 14% of patients in New York, USA (2-3).

In this subset of critically ill patients, multiple organ failure was seen, including cardiac dysfunction and decreased vascular tone, leading to hypotension. Risk factors for development of complications of Covid-19 include older age (e.g., >65 years), prior cardiovascular disease, chronic lung disease, hypertension, diabetes, and obesity (2, 3).

The cardiovascular complications of COVID-19 deserve especial attention. Myocardial injury, usually defined as increased troponin levels, with or without additional electrocardiographic or echocardiographic data suggesting cardiac damage, has been associated with adverse outcomes (4-8). The etiology of troponin elevation remains unclear in most cases - it may be due to either coronary plaque destabilization and acute myocardial infarction, myocardial blood flow supply/demand imbalance, microvascular ischemia, sepsis-related troponin increases, or may occur as a result of myocarditis caused by inflammatory cytokines or by the virus itself (6). As SARS-CoV-2 contaminates the host cells by means of the transmembrane angiotensin-converting enzyme (ACE)-II receptor, which is expressed in endothelial cells, there is a substrate for a direct pathogenic effect of the virus, as described by Varga et al (9), who found evidence of direct viral infection of endothelial cells and diffuse endothelial inflammation.

Recently, thrombotic complications of COVID-19, including deep vein thrombosis, pulmonary thromboembolism, peripheral and central arterial occlusions (11, 12) became widely recognized, such that the disease has been considered a syndrome of hyperinflammation, hypercoagulability and immunothrombosis. Platelet hyper-reactivity, hypercoagulability, hypofibrinolysis, complement overactivation, likely lead to a state of COVID-induced coagulopathy (13). D-dimer and prothrombin time are prognostic markers of adverse outcomes in COVID-19. (14, 15). Elevated D-dimer values at hospital admission and during further disease progression may reflect COVID-19-induced pulmonary inflammation with activation of platelets and blood coagulation (15).

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The understanding of the pathophysiology of cardiovascular complications in COVID-19 is a key to the implementation of therapeutic measures, which may offer additional benefits, mainly for patients with severe disease, such as anticoagulants. Continued research and data reporting, which have been performed with great effort, concomitantly to the fight against the disease, are fundamental in the progress of this knowledge.

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