Tomographic Findings and Thrombogenic Effects of COVID-19

Since December 2019, infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has progressively affected the populations of many countries around the world [1]. CT has become an important auxiliary tool in the management of this disease, used to confirm pulmonary involvement, identify complications, and define the prognosis [1]. Among the various publications on the topic, the report by Bao et al [1] titled “Coronavirus Disease 2019 (COVID-19) CT Findings: A Systematic Review and Meta-Analysis” caught our attention. We would like to discuss the contribution of the thrombogenic effects of the virus to tomographic findings and ask the authors if pulmonary thromboembolism was considered in their study.

Ground-glass opacities and areas of consolidation are commonly found on chest CT, not specific for any particular pathology, occurring in different types of viral pneumonia because of diffuse alveolar edema and interstitial inflammation [1]. In consequence, pulmonary involvement in coronavirus disease 2019 or in other coronavirus diseases, including severe acute respiratory syndrome and Middle East respiratory syndrome, can appear similar on CT [1]. However, in the current scenario, these findings in the peripheral and bilateral distributions are the hallmark of identification of lung involvement by the Sars-CoV-2, especially when located in the lower lobes [1].

Another critical point, also identified in recent months, is the predisposition to venous and arterial thrombotic complications [2], described in patients admitted to intensive care units and in general wards, even when using prophylactic anticoagulants [3]. Several studies have observed a correlation between coagulopathy and SARS-CoV-2 infection [3], with cases of deep vein thrombosis, thrombosis of the extracorporeal circulation circuits, pulmonary thromboembolism, and even arterial thrombosis being reported [3]. We have some cases of thromboembolism related to confirmed coronavirus infection in our institutions. Within this framework, different factors may be related to this condition, such as direct cellular injury by the virus, excessive inflammatory response, hypoxia, immobilization, and diffuse intravascular coagulation [2].

When a pathogen enters an organism, the inflammatory response is activated, stimulating defense cells and the coagulation cascade to prevent the spread of microorganisms throughout the organs and tissues [4]. Infection with SARS-CoV-2 stimulates a procoagulant activity related to inflammatory mechanisms through increased levels of fibrinogen, thrombin generation, plasminogen activator inhibitor-1, and direct endothelial injury that exposes the tissue factor, which leads to the activation of the coagulation cascade, as well as a reduced level of urokinase-type plasminogen activator [4]. In this respect, chest tomographic findings are remarkable, because arterial and venous vaso-occlusive diseases in the lungs can be seen as ground-glass opacities or areas of consolidation, representing pulmonary infarction with or without hemorrhage into the airspaces on histopathology [5].

In conclusion, it is possible to consider that findings on chest CT may represent not only direct lung involvement in the airways by the virus itself, as previously described. It is also probable that thrombogenic and embolic events in large vessels, identified on angiotomography, and in the microvasculature, which tomography cannot directly identify, may be related.

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