Original Research Paper

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Clinical Research

ENDOTHELITIS AND SARS-COV-2 INFECTION

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ABSTRACT The direct damage to the alveolar-capillary membrane by the Coronavirus determines a thromboembolic tendency. Monitoring through the blood dosage of D-Dimer and the pharmacoprophylaxis with low molecular weight heparin appear to be correct clinical attitudes in reducing morbidity and mortality.

KEYWORDS : SARS-CoV-2 Syndrome, Thromboembolism, D-Dimer test, Low molecular weight heparin.

"There are in fact two things, science and opinion; the former begets knowledge, the latter ignorance". Hippocrates. The clinical approach in medical practice, obtained through the epicrisis processes, has also gradually evolved in the light of new technological and instrumental acquisitions. The significance of the collection and analysis of clinical data fits well into this philosophical-scientific attitude. Then, learning converts to memory. Incident or necessity? Timeless commitment of science yet science is aseptic. SARS-CoV-2 pandemic is proving to be, for clinical researchers, a hotbed of pathophysiology new acquisitions. Specifically, in the field of vascular and respiratory pathophisiology. It has now been established that the endothelial damage caused by Coronavirus infection[1] is one of the anatomic, pathological and clinical bases for the appearance of a severe syndrome. So one of the three axioms of the Virchow triad - in addition to flow turbulence and hypercoagulable condition - is certainly involved in the determinism of thromboembolism regrettably so frequent in SARS-CoV-2 Syndrome. From December/1/2020 to March/31/2021 we have hospitalized in our Department twenty-five (25) patients (all not yet vaccinated) - with an average age of 72 years and 3 months - which had central thrombotic phenomena (deep venous circulation) and/or peripheral ones in the presence of Coronavirus infection. All patients had a high blood dose of D-Dimer, higher than 500 ng/ml (cut-off point). This condition is associated with the laboratory data of increased Fibrinogen degradation products - D-Dimer - between 4 to 9 times the standard. Fourteen (14) patients were female and eleven (11) were male. Peripheral phenomena could be limited to dischrome or cyanosis conditions at the extremities or petechial skin rashes. Eighteen (18) of the patients (72%) have then evidenced, in the follow-up, a persistence of the increase in the haematic values to cargo of the D-Dimer; six months after being negative to molecular swab, three (3) patients still had values above 500 ng/ml. For ten (10) of the patients (40%) the severe clinical onset occurred in conjunction with a pulmonary embolism condition. Fourteen (14) patients had a CT-Scan of bilateral interstitial pneumonia. The death rate was 16% (4 patients, 3 male and one female). The small number of cases does not allow statistical evaluations. We therefore focused our attention, in this sample of patients, on the blood control of D-Dimer. It is, as mentioned, the degradation product of Fibrinogen. The low blood values are due to a certain absence of thrombophilia or in any case of ongoing thrombophilia, while high values are suggestive (but not absolutely certain) for a condition of thrombosis in action. This goes for both the arterial and venous sides. In the absence of history compatible with recent trauma, including surgery, at patient's expense, the finding of a high D-Dimer value lays with good accuracy for a coagulative problem in his/her vascular system. Coronavirus-induced lesion at the alveolar-capillary membrane [2], with phenomena of leakage, confirms that the endothelial damage is the histopathological expression of SARS-CoV-2 Syndrome. In view of these clinical aspects, the early use of Enoxaparin at high doses, gradually scaled but retained until normalization of D-Dimer values, may have a preventive significance in reducing the onset of full-blow damage against the vascular endothelium [3]. Looks like endothelial cell damage is the cause of the inflammatory cascade and the subsequent formation of clots, with the mediation of ACE2 receptors. There are clinical data that testify of a negative conditioning on survival in the severe forms of SARS-CoV-2 Syndrome in the presence of high D-Dimer values [3,4]. SARS-CoV-2 infects the host using the ACE2 receptor: this receptor is expressed not only at the level of the lungs but also of the heart, kidney and bowel. ACE2 receptors are also expressed at endothelial level.

Through post-mortem in vitro analysis endothelial involvement is confirmed at the level of the vascular bed of various organs. Electron microscopy highlights viral inclusions within endothelial structures and infiltration of immune cells inside the vascular bed [1]. The pathogenic hypothesis is that the recruitment of immune cells and the direct damage caused by intraendothelial viral replications can underlie widespread microcirculation dysfunction. D-Dimer levels were more likely to be abnormal in severely and critically ill patients compared with mild and ordinary cases, while the D-Dimer levels of patients who had died were significantly higher than those of surviving patients according to the results of the lab tests. A D-Dimer value of 2025 ng/ml [5] could be most sensitive cutoff for a prognosis of death. In addition, we find that patients with advanced age, male gender, dyspnea symptoms and some underlying diseases have a higher D-Dimer value. Then, D-Dimer is related to the clinical classification. Can high D-Dimer data be a predictive marker of lower survival or higher incidence of thromboembolism in SARS-CoV-2 Syndrome, determining a coded therapeutic anticoagulant attitude? It is therefore now clear that a systemic coagulopathy condition in SARS-CoV-2 infection must be recognized immediately, without ever forgetting how a powerful and early therapeutic attitude leads, almost invariably, to an improved clinical outcome. The humility of the clinical researcher lies in the freedom to want to understand, without prejudice. In front of a new pathological condition, the process of understanding can be slow and even difficult, but always aimed at the goal of a positive relapse in terms of diagnosis and therapy. From the time of Galileo the intellectual approach is the same. Science becomes awareness. "If there is love for man, there will also be love for science". Hippocrates

Conflict of Interest

The Author declare that there is no conflict of interest .

REFERENCES

1. Varga Z, Flammer AJ, Steiger P, et al. (2020) Endothelial cell infection and endothelitis in COVID-19. Lancet Correspondence Vol 395: Issue 10234, P1417-1418.
2. Gavrilaki E, Anyfanti P, Gavrilaki M, et al. (2020) Endothelial dysfunction in COVID-19: Lessons learned from Coronavirus. Curr Hypertens Rep Aug 27; 22(9):63.
3. Willyard C (2020) Coronavirus blood-clot mystery intensifies.Nature 581:250.
4. Klok FA, Kruip MJHA, van der Meer NJM, et al. (2020) Incidence of thrombotic complications in critically ill ICU patients with COVID-19. Thrombosis Research 191: 145-147.
5. Zhang L, Yan X, Fan Q, et al. (2020) D-dimer levels on admission to predict in hospital mortality in patients with Covid-19. J Thromb Haemost 18: 1324-1329.