

Case Report

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The Endothelium as a Place of Virus Damage

Giorgio Bolla

Community Hospital, "Relaxxi"–Noale (Venice), Italy

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 use of pharmacoprophylaxis with low molecular weight heparin appear to be correct clinical attitudes in reducing morbidity and mortality.

*Corresponding author

Giorgio Bolla, Community Hospital, "Relaxxi"–Noale (Venice), Italy.

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Case Report

It's now clear that, in SARS-CoV-2 syndrome, a condition of systemic coagulopathy can take different meanings.

We report a clinical condition arrived to our attention. Conversely, we have analysed the dedicated Literature. Female patient, age 68, hospitalized for orthopaedic problems – and carrier of severe arthritis – is found to be positive to nasopharyngeal swab for SARS-CoV-2. During the period of positivity to the swabs the patient does not develop the typical bilateral interstitial pneumonia and never presents dyspnea. The only objective clinical data is the appearance of symmetrical cyanosis at the forefoot. This condition is associated with the laboratory data of increased fibrinogen degradation products – D-Dimer – between 4 to 9 times the standard (less than 500 ng/ml) throughout the period of positivity to the swabs. An arterial and venous Colour Echo Doppler examination of the lower limbs does not demonstrate thrombotic events in the explored districts. Therapy with Enoxaparin 40 mg. BD is established. Once achieved the negativity to the swab also the clinical picture of the forefoot is resolved – after a week -, while now after 40 days from this finding the value of D-Dimer is not within the normal range even if in constant decrease: it's currently 1,5 times greater. After 20 days from the onset of the negativity to the swab, the dosage of enoxaparin was reduced to 40 mg. OD. We believe that this treatment will continue, in the absence of clinical events, up to a month after the D-Dimer value has returned to the normal range.

Discussion

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, with phenomena of leakage, confirms that the endothelial damage is the histopathological expression of SARS-CoV-2 Syndrome [1,2]. 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 could be most sensitive cutoff for a prognosis of death [5]. 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.

Conflict of Interest

The author declare that there is no conflict of interest

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