Clarion Journal of Infectious Diseases and Therapy [JIDT]

Volume 2020 Issue 01

Case Report

The Clinical Expression of the Coagulopathy in Case of SARS-Cov-2

Giorgio Bolla*

Clinical Director – "Relaxxi" Hospital Noale (Venice-Italy)

Received: September 12, 2020; **Accepted:** September 18, 2020; **Published:** September 22, 2020

Citation: Bolla G (2020) The Clinical Expression of the Coagulopathy in Case of SARS-Cov-2. *Int J Infect Dis ther* 01(02): 10–11.

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 70, 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 (≤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 yet 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.

The Literature indicates the possible genesis on a vascular basis, with formation of thrombi in the pulmonary vessels, of viral pneumonia [1]. The appearance of pulmonary thromboembolism is also involved [2]. This condition of hypercoagulability, demonstrated by the elevated D-dimer, is now mentioned in several scientific papers [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 [1]. There are clinical data that lay for a negative conditioning on survival in the severe forms of SARS-CoV-2 syndrome in the presence of high D-dimer values [3].

The clinical case presented by us, where a dichotomy between the laboratory data and the modest clinical expression is evident, can lead to considerations:

- Are there in some subjects local molecular protective factors at the level of the pulmonary vessels that allow the patient with SARS-CoV-2 to avoid the complete clinical expression of the syndrome?
- Can be high D-dimer data a predictive marker of lower survival or higher incidence of thromboembolism in SARS-CoV-2 syndrome, determining a coded therapeutic anticoagulant attitude?

Further Studies will clarify these aspects better.

References

1. Willyard C (2020) Coronavirus blood-clot mystery intensifies. *Nature* 581: 250. [View]
2. 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. [View]
3. 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. [View]

*Corresponding author: Giorgio Bolla, Clinical Director – "Relaxxi" Hospital Noale, Venice-Italy;

Website: www.giorgiobolla.mnamon.it;

Email: giorgio.bolla@libero.it