

Low level anti-factor Xa activity sill efficient in decreasing D-dimer in COVID-19 patients

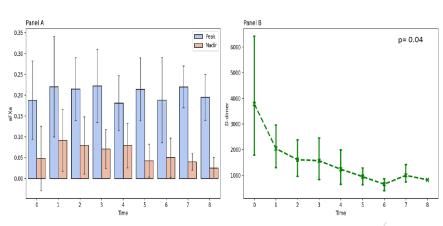
M.Bonifazi1, E. Sterchele1, M. Lucenteforte1, P. Simili1, G. Zuanetti1, S. Roli1, E. Chiodaroli1, P. Formenti1, L. Bolgiaghi1, S. Coppola1, D. Chiumello1

1. Department of Anesthesiology and Intensive Care, ASST Santi e Paolo Hospital, University of Milan, Milan, Italy

Introduction: COVID-19 patients show a pro-coagulant profile, thus a high thrombotic risk associated with inflammation1. Several case series showed evidence of severe endothelial injury and microvascular thrombosis. We aim to study the role of LBWHs anticoagulation through the monitoring of Anti-factor Xa (AFXa) in COVID-19 patients.

Methods:Twenty-six COVID-19 patients receiving enoxaparin 4000 UI x 2/die as anticoagulation therapy from December 1st till April 26th were enrolled. Day 0 was the 1st day in which they received enoxaparin Laboratory tests (blood count, coagulation, renal and liver function) and AFXa dosing (HemosIL, Liquid Anti-Xa) 1h before the administration of enoxaparin and 4h after were performed. every 72 hours and so on until patient discharge, even if enoxaparin dose was changed.

Results: As shown in Panel A, Fig 1 the AFXa peak did not change significantly throughout each time point measurement as well as the AFXa nadir (p= 08 and 0.65 respectively). D-dimer, however, progressively, and significantly decreased over time (p= 0.04). Its change was associated with nadir AFXa steady levels (mean value 0.08 ± 0.07 UI/mL) and time and not to peak AFXa levels (mean value 0.2 ± 0.1 UI/mL). During the ICU stay mean creatinine was 0.7 ± 0.3 mg/dL, mean INR was 1.08 ± 0.18 , mean aPTT ratio 0.98 ± 0.25 . **Conclusion:** Anticoagulation levels of AFXa were never reached regardless of the LBWHs anticoagulation dose. Nonetheless D-dimer decreased and was associated with low but steady level of AFXa nadir. It is possible that a prophylactic level of AFXa could be effective in the treatment of microvascular thrombosis considering the pro-inflammatory aspect of the disease. Further research is needed to understand the underlying mechanism.



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