Cytokine storm induced coagulopathy in septic shock and critical Covid-19: head-to-head comparison

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Background: Septic shock generates an important inflammatory reaction, endothelial activation and a procoagulant state leading to microvascular thrombosis and subsequent organ impairment [1].
Similarly, a severe inflammatory reaction and a coagulopathy with pulmonary micro-thrombosis eventually leading to acute lung injury, is a typical feature of critical form of Coronavirus disease 2019 (Covid-19) [2].
Our aim was to compare coagulation, platelet activation and platelets-neutrophils interplay between control, septic shock and critical Covid-19 patients.
Methods/Materials: A total of 118 patients were included in our prospective, monocentric, observational study between February 2019 and June 2020. Septic shock (n=48) and Covid-19 (n=22) patients were consecutively included at admission in our ICU department. Control patients (n=48) with matched gender and co-morbidities were recruited at central lab consultation.
Results: Septic shock patients had worse severity scores due to multiple organ failure (assessed by APACHE II and SOFA score) whereas Covid-19 patients had more severe respiratory failure and a longer ICU length-of-stay (Table 1).
At the time of inclusion, CRP and lymphocyte count were comparable between septic shock and Covid-19 patients. White cell count ad neutrophil count was higher for septic shock patients.
Analysis of coagulation showed a prolonged INR, TT and aPTT in septic shock although only INR was prolonged in Covid-19. Thrombin antithrombin complex (TATc) formation was similar in both pathologies, whereas consumption of antithrombin III (ATIII) and D-dimers formation was more pronounced in septic shock.
Platelet count was lower in septic shock and platelet activation, assessed via plasmatic levels of soluble P-selectin (sCD62P) and Trem-like transcript 1 (sTLT-1), was more important in septic shock.
Neutrophil activation and NETosis, evaluated by levels of circulating myeloperoxidase (MPO) and citrullinated histone 3 (H3-Cit), was similarly increased in both groups (Figure 1).
Conclusions: This study confirmed an activation of coagulation cascade, platelet activation and NETosis in both septic shock and critical Covid-19, compared with control patients. Importantly, the extent of these changes was similar or less pronounced in critical COVID-19 compared with septic shock.

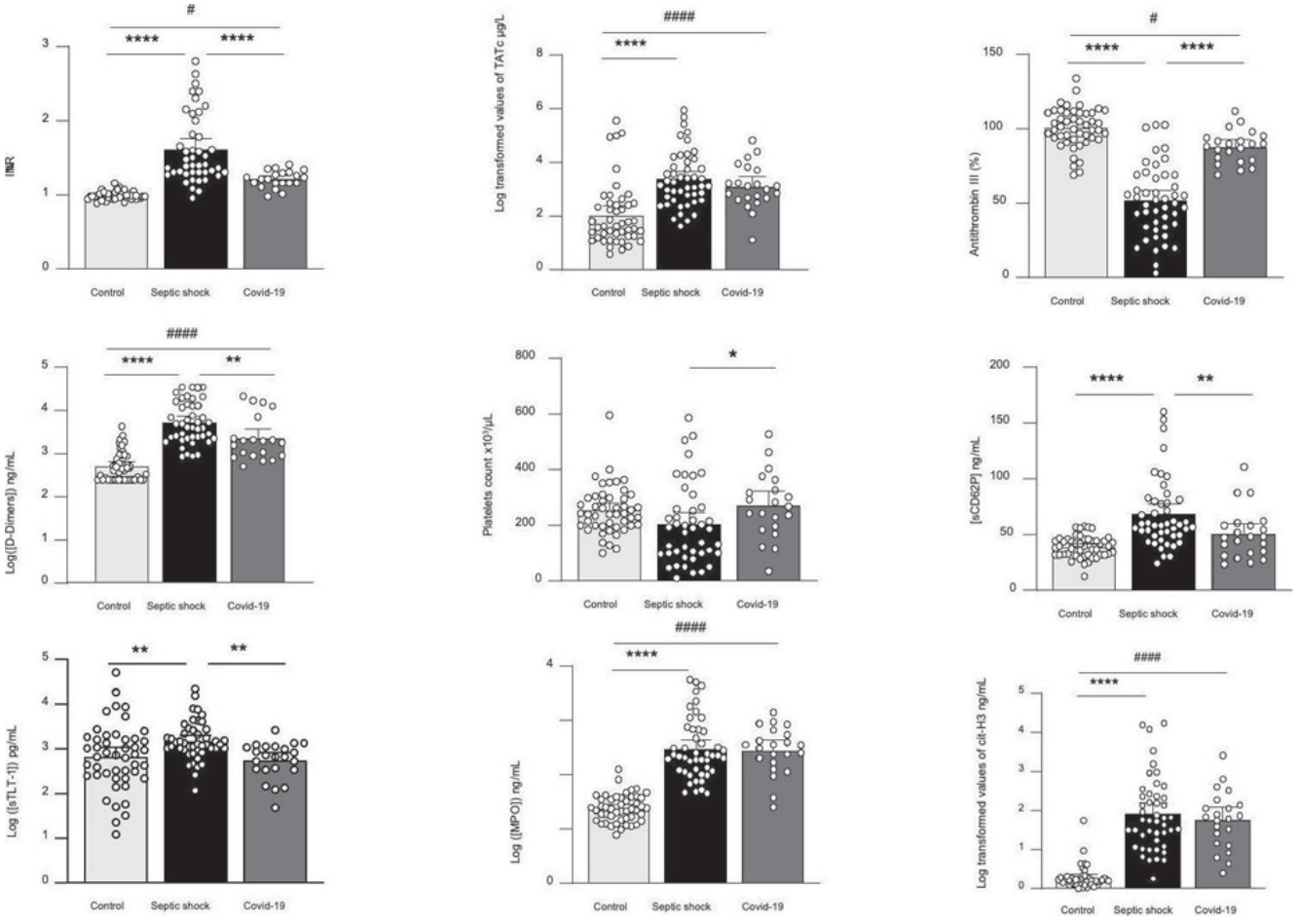

Figure 1