Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Archives of Cardiovascular Disease Supplements 16 (2021) 182—183

Available online at ScienceDirect www.sciencedirect.com

Elsevier Masson France EM|consulte www.em-consulte.com/en

02 — Thrombosis, Hemostasis

342

Head-to-head comparison of cytokines storm-coagulopathy in septic shock and COVID-19

J. De Poortere[1,*], M. Dechamps[1,2], P.F. Laterre[3], M. Octave[1], A. Ginion[1], V. Robaux[1], L. Pirotton[1], J. Bodart[1], L. Gerard[1,3], D. Gruson[4], M.A. Van Dievoet[4], J. Douxfils[5], M. Derive[6], L. Gatto[7], M. Martin[7], C. Bouzin[1], D. Castanares-Zapatero[1,3], L. Bertrand[1], S. Horman[1], C. Beauloye[1,8]

[1] *Institut de recherche expérimentale et clinique (IREC), CARD, Université Catholique de Louvain, Brussels, Belgium*

[2] *Cardiovascular Intensive Care, Cliniques Universitaires St Luc, Brussels, Belgium*

[3] *Intensive Care, Cliniques Universitaires St Luc, Brussels, Belgium*

[4] *Clinical Biology, Brussels, Cliniques Universitaires St Luc, Brussels, Belgium*

[5] *Namur Research Institute for Life Sciences (Narilis), Department of Pharmacy, Université de Namur, Namur, Belgium*

[6] *Inotrem SA, Vandœuvre-Lès-Nancy, France*

[7] *Institut de Duve (DDUV), Université catholique de Louvain, Brussels, Belgium*

[8] *Cardiology, Cliniques Universitaires St Luc, Brussels, Belgium*

* *Corresponding author.*

E-mail address: julien.depoortere@uclouvain.be (J. De Poortere)

Introduction Host immune response to the coronavirus disease 2019 (COVID-19) is variable and can induce a dysregulated inflammatory response associated with venous and arterial thrombosis called COVID-19 associated coagulopathy (CAC). During septic shock, inflammatory reaction generates endothelial activation and procoagulant state with microvascular thrombi inducing disseminated intravascular coagulation (DIC). Although CAC and DIC induce altered coagulation responses, their clinical outcomes are different.

Objective We investigated and compared coagulopathy between septic shock and critical COVID-19 patients.

Method Septic shock patients were diagnosed following the Survival Sepsis Campaign guidelines. COVID-19 patients were admitted in intensive care unit (ICU) for severe acute respiratory distress syndrome. Both were included in the study within 2 days after admission. Biomarkers were measured by ELISA from patient's plasma.

Results We observed an increase in vWF and TFPI in both septic and COVID-19 patients compared to controls, highlighting endothelial damage. Interestingly, circulating TF was only elevated in COVID-19 patients. Platelet activation differed between the two cohorts of patients. P-selectin and TLT-1 were specifically heightened in septic shock whereas CD40L was only augmented in COVID-19. Coagulation markers were increased in a disease-dependent way, with PAI-1, tPA and D-Dimers higher in septic shock and fibrinogen level, higher in COVID-19.

Discussion COVID-19 patients had longer length-of-stay with more pronounced respiratory failure. This strong lung disruption overtime induced plasmatic TF release with sustained inflammatory response characterized by sCD40L and fibrinogen secretion. Given the similarities between COVID-19 and septic shock regarding fibrinolysis and coagulation, but not platelet activation, endothelium seems to play a central role in COVID-19 and might explain the differences between CAC and DIC.

Disclosure of interest The authors declare that they have no competing interest.

https://doi.org/10.1016/j.acvdsp.2021.04.089

332

Acetyl-CoA carboxylase inhibition alters tubulin acetylation and aggregation in thrombin-stimulated platelets

M. Octave[1,*], L. Pirotton[1], A. Ginion[1], V. Robaux[1], S. Lepropre[1], S. Kautbally[1], V. Darley-Usmar[2], J. Ambroise[3], B. Guigas[4], M. Giera[4], M. Foretz[5], L. Bertrand[1], C. Beauloye[1], S. Horman[1]

[1] *Institut de Recherche Expérimentale et Clinique, Pôle de Recherche Cardiovasculaire, Université catholique de Louvain, Bruxelles, Belgium*

[2] *Center for Free Radical Biology, Department of Pathology, UAB Mitochondrial Medicine Laboratory, University of Alabama at Birmingham, Birmingham, USA*

[3] *Institut de Recherche Expérimentale et Clinique, Centre de technologies moléculaires appliquées, Université Catholique de Louvain, Bruxelles, Belgium*

[4] *Leiden university medical center, Leiden, Netherlands*

[5] *Institut Cochin, INSERM, U1016-CNRS UMR8104, Université Paris Descartes, Paris, France*

* *Corresponding author.*

E-mail address: marie.octave@uclouvain.be (M. Octave)

Introduction Acetyl-CoA carboxylase (ACC), the first enzyme regulating lipid synthesis, promotes thrombus formation by increasing platelet phospholipid content. Inhibition of its activity decreases lipogenesis and increases the content in acetyl-CoA which can serve as a substrate for protein acetylation. This posttranslational modification plays a key role in the regulation of platelet aggregation, via tubulin acetylation.