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Transient endothelial injury and release of lupus anticoagulant in COVID-19



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Introduction Coronavirus disease 2019 (COVID-19) has been described as an endothelial disease associated with a procoagulant state and a high prevalence of lupus anticoagulant (LA). No study has so far evaluated the persistence of endothelial injury after recovery.

Purpose We report the results of a systematic biologic assessment more than 12 weeks after the acute phase of COVID-19.

Methods Patients hospitalized for COVID-19 at Strasbourg university hospital, France, and tested positive for LA were included in the microparticles in COVID-19 (MICO) study. During the prospective follow-up, blood samples were obtained at least 12 weeks after COVID-19 diagnosis.

Results Between March 3 and May 5, 2020, 56 COVID-19 patients with positive LA were included in the study. Five patients were excluded from the analysis because of direct oral anticoagulant treatment at the time of follow-up. A total of 51 patients were included in the final analysis. The mean age was 61 years. During the acute phase of COVID-19, 38 patients (74.5%) required mechanical ventilation, 10 patients (19.7%) presented a venous thrombotic event and mean von Willebrand factor antigen (vWF:Ag) level was 409.5%. Follow-up visit was performed at a median of 144 (interquartile range 129–179) days after COVID-19 diagnosis. LA detection was positive only in three patients (5.9%) and mean level of vWF:Ag was 158.0% at the time of follow-up. No thrombotic event was observed during the follow-up phase (Fig. 1, Table 1).

Conclusions We showed disappearance of LA in a large majority of patients and a drastic decrease of vWF:Ag levels, clinically translated by the absence of thrombosis event during the follow-up. Our results suggest that endothelial dysfunction is transient in COVID-19 patients and therefore associated to a potential temporary and limited pathophysiological effect.

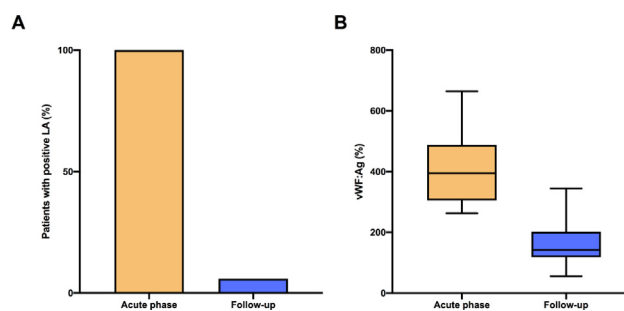


Fig. 1 Transient lupus anticoagulant positivity and high vWF:Ag levels during acute COVID-19.

Table 1 Characteristics of the study population.

Characteristics of the study population (n = 51)		
Characteristics	Acute phase	Follow-up
Age – years	61 ± 13	
Male – n (%)	39 (76.5)	
Body mass index – kg/m ²	27.3 ± 3.8	
Comorbidities		
Hypertension	24 (47.1)	
Dyslipidemia	15 (29.4)	
Diabetes	7 (13.7)	
Coronary artery disease	5 (9.8)	
History of venous thromboembolism	3 (5.9)	
Chronic respiratory disease	6 (11.8)	
COVID-19 outcomes		
Acute pulmonary embolism	8 (15.7)	0 (0.0)
Deep vein thrombosis	3 (5.9)	0 (0.0)
Transfer to intensive care unit	41 (80.4)	0 (0.0)
Mechanic ventilation	38 (74.5)	0 (0.0)
CRP – mg/L	175.6 ± 106.6	5.2 ± 2.8
Fibrinogen – g/L	7.5 ± 1.6	3.7 ± 0.9
aPTT – %	1.4 ± 0.5	1.1 ± 0.3
D-Dimer – ng/mL	3841 ± 4375	414 ± 219
LA detection – n (%)	51 (100)	3 (5.9)
vWF:Ag – %	409.5 ± 107.5	158.0 ± 59.2

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

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Impact of cardiac and acute kidney injury on COVID-19 in-hospital mortality



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Background and aim The severity of Coronavirus Disease 2019 (COVID-19) is a multifactorial condition. Cardiac and acute kidney injury (AKI) are two complications commonly reported in severe forms of COVID-19. We aimed to investigate the effect of these two complications on the COVID-19 in-hospital mortality.

Materials and methods This is a prospective study, including 120 severe cases of COVID-19, admitted at the university hospital of Blida. Troponin was assessed by an immuno-fluoroassay method. AKI was defined according to the KDIGO-2012 guidelines. The association with in-hospital mortality was assessed using the Kaplan–Meier survival curve, proportional Cox regression analyses and the receiver operating characteristic curve.

Results Cardiac and acute kidney injury were very common, occurring in 19% and 25% of patients. When analyzing survival, both were significantly associated with in-hospital mortality ($P_{\text{LogRank}} < 0.0001$). A cutoff value of 9.6 ng/mL for troponin and 13.9 mg/L for creatinine could predict poor prognosis with a sensitivity of 73% and 67%, and a specificity of 62% and 64%, respectively. Hazard ratios were (HR=3.5, 95% CI [1.7–7.3], $P=0.001$ and HR=3.14, 95% CI [1.6–6.1], $P=0.001$) for troponin cutoff and AKI respectively.

Conclusion This study demonstrates the high frequency of cardiac and acute kidney injury in severe COVID-19 patients and provides further evidence of their potential link to poor short-term prognosis.

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

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Endothelial dysfunction is the key of long COVID-19 symptoms: The results of TUN-EndCOV study



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Background The COVID-19 disease is a multisystem disease due to in part to the vascular endothelium injury. Lasting effects and long-term sequelae could persist after the infection and may be due to persistent endothelial dysfunction.

Purpose Our study focused on the study of endothelial function measurement by digital thermal monitoring (DTM) of endothelial quality index with E4 diagnosis Polymath in a large cohort of long COVID-19 patients to determine whether long COVID-19 symptoms are due to endothelial dysfunction.

Methods This is a prospective multicenter longitudinal observational cohort study. Endothelial function was evaluated with "E4-Diagnose" Polymath Tunisia based on the Endothelium Quality Index (EQI). A complete echocardiographic evaluation analysis was performed. Primary outcomes were defined as the occurrence of long COVID-19 symptoms in patients with endothelial dysfunction measured by EQI.

Results A total of 798 patients were included in this study. Patients were included at an average time of 68.93 ± 43.1 days. The mean EQI was 2.02 ± 0.99 [0–5]. A total of 397 (49.7%) patients had poor or very poor EQI and 211 (26.4%) patients had very poor EQI. The median age was 49.94 ± 14.2 (18–80) years. A total of 618 patients (77.4%) had long COVID-19 symptoms. Patients with long COVID-19 symptoms had a reduced EQI (1.99 ± 0.97 vs. 2.09 ± 1.05 , $P=0.24$). Among long COVID-19 symptoms, fatigue was the most common symptom reported in 42.2%. Fatigue and chest pain were significantly associated to the endothelial dysfunction ($P=0.04$ and 0.001 respectively). Patients with chest pain had significantly lower EQI (1.74 ± 1.0 vs. 2.09 ± 0.9 , $P \leq 10^{-3}$) and LVGLS (-16.35 ± 3.0 vs. -17.16 ± 2.5 , $P=0.04$).

Conclusion Long COVID-19 symptoms specifically chest pain and fatigue are due to persistent poor endothelial quality index. These findings allow a better care of patients with long COVID-19 symptoms.

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

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Predictors of thromboembolic events in COVID-19 ambulatory patients



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Introduction Unlikely COVID-19 hospitalized patients, there are not clear data about the incidence and the predictors of arterial and venous thromboembolic events in COVID-19 outpatients patients.

Objectives To determine the incidence of TE in COVID-19 ambulatory setting.

Patients and methods We conducted an observational study in our study including COVID-19 ambulatory patients and we analysed the predictors of TE events at 30 days.

Results We included 2089 patients with a mean age of 43 ± 16 years. The incidence of primary outcomes was 1.6% and the incidence of venous and arterial TE complications was 0.9%. The predictors of arterial and venous TE complications were hormonal contraception (OR=23), moderate clinical presentation (OR=3.5), recent surgery or miscarriage during the last month before COVID-19 infection (OR=9.2) and COVID-19 signs on CT scan (OR=4.9). While physical activity proved to be a protective factor.