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## Editorial

# COVID-19 and ischemic stroke: Should we systematically look for lupus anticoagulant and antiphospholipid antibodies?



Coronavirus disease 2019 (COVID-19) due to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has spread rapidly throughout the globe [1]. The spectrum of disease is broad ranging from asymptomatic cases to multi-organs failures. The main impairment is respiratory with: dry cough, dyspnea or acute respiratory distress syndrome. However, neurological impairments are also described including: headache, hyposmia, hypogeusia, dizziness, impaired consciousness, seizure, encephalopathy, neuromuscular injury or acute cerebrovascular disease [2]. In a Chinese retrospective study of 214 COVID-19 patients, 78 (36.4%) had neurologic manifestations [3]. Incidence of ischemic stroke in context of COVID-19 varies according preliminary studies from 2.5 to 6.4% [3–6].

Many studies reporting that the behavior of clotting variables and platelet count in COVID-19 patients suggest a hyper-coagulation state associated with a significant increased risk of thromboembolic events [7]. A recent meta-analysis suggests that elevated plasma levels of D-dimers constituted an independent biomarker for poor prognosis in COVID-19 patients [8]. Pathophysiology of this hyper-coagulation state remain incompletely understood and probably multifactorial favored by systemic inflammation and cytokine storm [7,9]. Some authors also suggest existence of a specific vasculopathy due to direct vascular endothelial cell infection by SARS-CoV-2 through the angiotensin converting enzyme 2 receptor [10].

Lupus anticoagulant (LAC) and anti-phospholipid antibodies (APAb) are known to increase thromboembolic risk and be associated with anti-phospholipid syndrome (APS) [11]. In COVID-19 patients little data are currently available [12–14]. However, little data available are interesting and should lead us to ask some questions. Indeed, in an article Zhang et al. [12] report 3 cases of ischemic stroke with presence of APAb represented by anti-cardiolipin (aCL) and anti- $\beta$ 2-glycoprotein I ( $\beta$ 2GPI) in COVID-19 patients without history of APS. In another article, Beyrouti et al. [13] report 5 cases of ischemic stroke with presence of LAC in COVID-19 patients without history of APS. Finally, in a French cohort study of 56 COVID-19 patients Harzallah et al. [14] report 25 positive cases for LAC and 5 positive cases for aCL or  $\beta$ 2GPI. Otherwise, in another

article Oxley et al. [15] report 5 cases of ischemic stroke in young patients without significant medical history. Unfortunately, LAC and APAb are not reported.

Acute infections are sometimes associated with transient positive LAC or APAb without clinical signs. In this case, anticoagulant therapy is usually not necessary [11]. However, detection of LAC with or without aCL or  $\beta$ 2GPI in symptomatic COVID-19 patients characterized by significant increased risk of thromboembolic events highlight the importance of an early detection for better therapeutic management. Implication of LAC or APAb in COVID-19 pathophysiology remain poorly understood. However, it could be involved and their pro-coagulant action could be involved in thromboembolic events due to COVID-19, in particular in ischemic stroke. From our point of view, more studies are necessary to elucidate roles of LAC and APAb in COVID-19. However, it may be interesting to look for LAC and APAb in our COVID-19 patients with thrombotic event or high D-Dimeres level to adapt anticoagulation level. Current data suggest use of prophylactic anticoagulation with low-molecular-weight-heparin in hospitalized COVID-19 patients [7]. Full intensity anticoagulation should not be used unless otherwise indicated such as positivity of LAC and/or APAb [11].

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### Author contributions

All authors affirm that they have contributed equally to the production of this manuscript.

### Disclosure of interest

The authors declare that they have no competing interests.

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