Endotheliopathy

Increasing evidence suggests that the adhesive ligand von Willebrand factor (VWF), which is synthesized in and released from endothelial cells, plays a paradoxical role in both facilitating local hemostasis at the site of injury and also propagating TBI-induced endotheliopathy and coagulopathy systemically [1].

Hernández-Fernández et al. presented cerebrovascular disease case incidence in hospitalized patients with SARS-CoV-2 infection. Patients were confirmed by microbiological/serological testing, or on chest CT semiology. Available data on comorbidity, laboratory parameters, treatment administered, neuroimaging, neuropathological studies and clinical evolution during hospitalization, measured by the modified Rankin scale, were analysed. A bivariate study was also designed to identify differences between ischaemic and haemorrhagic subtypes. A statistical model of binary logistic regression and sensitivity analysis was designed to study the influence of independent variables over prognosis. In our centre, there were 1683 admissions of patients with COVID-19 over 50 days, of which 23 (1.4%) developed cerebrovascular disease. Within this group of patients, cerebral and chest CT scans were performed in all cases, and MRI in six (26.1%). Histological samples were obtained in 6/23 cases (two brain biopsies, and four arterial thrombi). Seventeen patients were classified as cerebral ischaemia (73.9%, with two arterial dissections), five as Intracerebral hemorrhage (21.7%), and one leukoencephalopathy of posterior reversible encephalopathy type. Haemorrhagic patients had higher ferritin levels at the time of stroke (1554.3 versus 519.2, P = 0.004). Ischaemic strokes were unexpectedly frequent in the vertebrobasilar territory (6/17, 35.3%). In the haemorrhagic group, a characteristic radiological pattern was identified showing subarachnoid haemorrhage, parieto-occipital leukoencephalopathy, microbleeds and single or multiple focal haematomas. Brain biopsies performed showed signs of thrombotic microangiopathy and endothelial injury, with no evidence of vasculitis or necrotizing encephalitis. The functional prognosis during the hospital period was unfavourable in 73.9% (17/23 modified Rankin scale 4-6), and age was the main predictive variable (odds ratio = 1.5; 95% confidence interval 1.012-2.225; P = 0.043). Our series shows cerebrovascular disease incidence of 1.4% in patients with COVID-19 with high morbidity and mortality. We describe pathological and radiological data consistent with thrombotic microangiopathy caused by endotheliopathy with a haemorrhagic predisposition [2].

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Xu X, Kozar R, Zhang J, Dong JF. Diverse Activities of von Willebrand Factor in Traumatic Brain Injury and Associated Coagulopathy [published online ahead of print, 2020 Sep 15]. J Thromb Haemost. 2020;10.1111/jth.15096. doi:10.1111/jth.15096
2)
Hernández-Fernández F, Valencia HS, Barbella-Aponte RA, et al. Cerebrovascular disease in patients with COVID-19: neuroimaging, histological and clinical description [published online ahead of print, 2020 Jul 9]. Brain. 2020;awaa239. doi:10.1093/brain/awaa239