Int. j. adv. multidisc. res. stud. 2022; 2(6):944-945

Received: 03-12-2022
Accepted: 10-12-2022

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

The etiology of SARS-CoV-2 associated juvenile ischemic stroke in COVID-19 is diverse

[1] Josef Finsterer, [2] Anna Bersano
[1] Neurology & Neurophysiology Center, Vienna, Austria
[2] Fondazione IRCCS Istituto Neurologico Carlo Besta, Milano, Italy

Corresponding Author: **Josef Finsterer**

We read with interest the article by Mozhdehipanah *et al*. about the etiology of juvenile, ischemic stroke in five patients with COVID-19 [1]. It was concluded that a causal relation between COVID-19 and stroke was not confirmed but cannot be excluded in the context of a systemic prothrombotic state [1]. The study is appealing but raises comments and concerns.

A limitation of the study is that imaging was carried out only with cerebral `computed tomography (CT) in patient-1, patient-2, and patient-3. Other cerebral complications of a SARS-CoV-2 infection, such as acute disseminated encephalo-myelitis (ADEM), posterior reversible encephalopathy syndrome (PRES), venous sinus thrombosis, VST, acute, hemorrhaghic, necrotising encephalitis (AHNE), immune encephalitis, reversible cerebral vasoconstriction syndrome (RCVS), or cerebral vasculitis can be more appropriately excluded by multimodal cerebral magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA).

It is not comprehensible why patient-1 was treated with unfractionated heparin intravenously [1]. This procedure is not in accordance with international guidelines for the treatment of ischemic stroke. The rationale for this procedure should be provided.

It is not comprehensible why patient-1, patient-2, and patient-5 were treated with chloroquine. It is meanwhile well-known that it is ineffective for COVID-19. Furthermore, chloroquine may exhibit various side effects which can be harmful for the individual and negative for the outcome.

Another point of discussion is why the anti-COVID-19 treatment with chloroquine in patient-2 was initiated without prior confirmation of the infection with appropriate methods.

Concerning patient-4 it is contradictory to characterise the patient as “healthy opiate addict” as opiate addiction is a disease according to the international classification of diseases (ICD).

We also wonder why patient-4 was diagnosed with ischemic stroke although cerebral MRI indicated a PRES [1]. Obviously the patient only received anti-seizure drugs (ASDs) but no anti-platelet therapy. We should know why the patient was included in the case series. The cause of intracerebral bleeding should be also provided.

Patient-4 had an enlarged left atrium and reduced systolic function suggesting atrial fibrillation (AF) [1]. Was AF recorded on ECG? Did the patient also present with heart failure?

Patient-1 obviously died from generalised cerebral edema but the cause of the edema was not provided [1]. Was the edema attributable to the stroke? The NIHSS score on admission was only 6. Why did the patient develop hydrocephalus? Were there any indications for a second cerebral disease? Did the patient undergo autopsy?

An information about the extra-cranial cerebral vessels should be provided. We should know if there were any indications for dissection, occlusion, or hemodynamically significant stenosis.

The information about the classical risk factors for stroke/embolism is partially lacking. How many of the five patients had arterial hypertension, diabetes, atrial fibrillation, hyperlipidemia, or were smokers.

According to the discussion, three patients had large vessel occlusion [1]. However, CT-angiography had been carried out only in patient-3. Thus, we wonder how the occlusion was diagnosed in the remaining two patients.

SARS-CoV-2 infections may not only be associated with hypercoagulability but also with hypo-coagulability [2] and thus a propensity for bleeding. Bleeding may not only be associated with arterial hypertension but also with immune thrombocytopenia [3], disseminated intravascular coagulation (DIC) syndrome [4], or reduced production of coagulation factors.

Overall, the study has several limitations, which challenge the results and their interpretation. These issues should be addressed not to draw unsupported conclusions.

Declarations

Ethical approval and consent to participate: Not applicable.

Consent for Publication: Not applicable.

Availability of data and material: All data reported are available from the corresponding author.

Competing interests: None.

Funding: None received.

Acknowledgements: None.

Author contribution: JF: design, literature search, discussion, first draft, critical comments, AB: literature search, discussion, critical comments, final approval.

Keywords: COVID-19, SARS-CoV-2, Outcome, Stroke, Hypercoagulability

References

1. Mozhdehipanah H, Paybast S, Mavandadi S. Association of COVID-19 Infection and Juvenile Stroke: A Case Series. Acta Neurol Taiwan. 2021; 30(1):27-34.
2. Kong R, Hutchinson N, Görlinger K. Hyper- and hypocoagulability in COVID-19 as assessed by thromboelastometry-two case reports. Korean J Anesthesiol. 2021; 74(4):350-354. Doi: 10.4097/kja.20327.
3. Petcu AE, Dumitru IM, Cernat RC, Șerban CI, Hangan A, Ghiță E, *et al*. Immune thrombocytopenic purpura secondary to COVID-19 - case report. Germs. 2021; 11(2):319-323. Doi: 10.18683/germs.2021.1268.
4. Vorobyev PA, Momot AP, Krasnova LS, Vorobiev AP, Talipov AK. Pathogenesis, diagnosis, prevention and treatment of disseminated intravascular coagulation syndrome in COVID-19 infection. Ter Arkh. 2020; 92(11):51-56.