Journal of Medical Case Reports and Reviews

Received 14 Jan 2021 | Revised 18 Jan 2021 | Accepted 17 Mar 2021 | Published Online 22 Mar 2021

JMCRR 04 (03), 832-833 (2021) ISSN (O) 2589-8655 | (P) 2589-8647

JMCRR

INCREASED LUMBAR PUNCTURE OPENING PRESSURE NOT NECESSARILY REFLECTS INCREASED INTRACRANIAL PRESSURE IN COVID-19

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Key words: SARS-CoV-2, COVID-19, headache, intra-cranial pressure, multi-system inflammatory syndrome

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Letter to the Editor

With interest we read the article by Baccarella et al. about two children (9yo male (patient-1), 6yo male (patient-2)) with multisystem inflammatory syndrome in childhood (MIS-C) manifesting with increased intracranial pressure(IICP) and regarded as a sequelae of a SARS-CoV-2 infection.[1] We have the following concerns.

The first shortcoming is that it remains questionable if the ICP was trly increased in patient-1. Patient-1 had headache and right abducens palsy but no papilledema, kinking of the optic nerve, or distension of the optic nerve sheath.[1] The diagnosis "IICP" was based solely on the clinical presentation and an increased lumbar puncture opening pressure (ILOP).[1] Headache is multi-causal and cranial nerve palsies in COVID-19 not solely reflect IICP. Furthermore, an ILOP not necessarily reflects an IICP. In a study of 34 patients with spinal muscular atrophy receiving nusinersen, the LOP was >20 cm H2O in 25 asymptomatic patients. Only in one patient with ILOP was there papilledema and evidence for IICP on imaging. There is also the discrepancy between IICP on imaging but normal LOP in patient-2,[1] why we should know the latency between cerebral MRI and spinal tap.

A second shortcoming is that the diagnosis COVID-19 in patient-1 was not substantiated by a PCR-test but only by antibody tests.[1] Presence of anti-SARS-CoV-2 antibodies does not necessarily indicate the time when the patient had been infected. Furthermore, it was not detailed if IgG- or IgM-antibodies or both were elevated in patient-1. Thus, it is conceivable that the clinical presentation in patient-1 was unrelated to the viral infection.

A further shortcoming is that patient-2 had received intravenous immunoglobulins (IVIG) prior to development of neurological deficits.[1] From IVIGs it is well-known that they increase the risk of thrombosis, including venous sinus thrombosis, and thus IICP.

A fourth shortcoming is that cytokine levels were neither measured in the CSF nor in the serum. From interleukin-(IL)17, for example, it is known that it is associated with idiopathic intracranial hypertension.

IICP in MIS-C is explained by SARS-CoV-2 associated coagulopathy, but no evidence for coagulopathy in the two index patients was provided.

Overall, the study has several limitations which should be met before drawing conclusions as those presented. There is poor evidence for IICP in patient-1 and a causal relation between presentation and SARS-CoV-2 has not been substantiated. Diagnosing MIS-C should not only rely on the clinical presentation but also on reliable laboratory biomarkers.

Declarations

Acknowledgement: none

Statement of ethics: was in accordance if ethical guidelines

Conflicts of interest: none

Funding sources: no funding was received

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

Informed consent: was obtained

The study was approved by the institutional review board

References:

1 Baccarella A, et al. Pediatr Neurol 2021;115:48-49.