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Letter to the Editor. Diagnosis of immune encephalitis requires a workup for specific antibodies and the exclusion of SARS-CoV-2 infection

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We read with interest Konopka et al's article about a 15-year-old male with autoimmune encephalitis (AIE) who was initially diagnosed with psychosis manifested by behavioral changes, delusions, auditory hallucinations, hyperactivity, insomnia, and headaches [1]. However, haloperidol and olanzapine were ineffective. The initial cerebral MRI showed T2/FLAIR hyperintensities in the basal ganglia and corona radiata bilaterally and the follow-up MRI additionally T2/FLAIR hyperintensities in the posterior limb of the left internal capsule [1]. The initial and follow-up cerebrospinal fluid (CSF) examination revealed pleocytosis and elevated protein [1]. GM1 and GM2 antibodies were positive. The patient benefited from methyl-prednisolone and intravenous immunoglobulins (IVIG) [1]. The work is compelling, but some points should be discussed.

The first point is that the index patient was not tested for extra – or intra-cellular AIE-associated antibodies. Because certain intracellular AIE-associated antibodies can indicate malignancy, it would have been imperative to test the patient for these antibodies. Additionally, it would have been crucial to screen the patient for malignancies or other diseases associated with AIE.

A second point is that the patient was not tested for SARS-CoV-2 infection using RT-PCR. Since the case apparently occurred during the pandemic, it would have been imperative to know whether the patient had COVID-19 or not.

A third point is that several findings on the clinical neurological examination remained unclear. We should know why the patient had decreased patella tendon reflexes. Was there evidence of radiculitis or polyneuropathy? Did the patient develop critically ill neuropathy or myopathy in the ICU? The patient was described as having upper limb weakness. What was the cause of upper extremity paraparesis?

A fourth point is that AIE can manifest with a broader range of clinical manifestations than is reflected in the statement that "AIE manifests with rapidly progressive

short memory deficits or cognitive impairments accompanied by multiple disorders related to the limbic system" [1]. In addition to these manifestations, AIE can manifest with impaired consciousness, disorientation, confusion, psychosis, epilepsy, movement disorder, speech disorder, headache, meningismus, fever, autonomic dysfunction, central apnea, coma and death.

A fifth point is that the specific white blood cell count/and the amount of CSF protein increase were not quantified. In order to assess the etiology of encephalitis, it would have been imperative to know the CSF cell count and cell types as well as the amount of CSF protein. Knowledge of these quantifications is crucial for follow-up studies and for assessing whether these parameters improved, remained unchanged, or increased.

A sixth point is that follow-up cerebral MRI was apparently performed without the administration of contrast medium. In order to be able to assess the extent and dynamics of cerebral inflammation, the administration of gadolinium would have been mandatory. Another limitation of imaging studies is that DWI, ADC, and SWI modalities were not used. Multimodal MRI would have been important to determine whether the described lesion represented cytotoxic edema, vasogenic edema, or gliosis.

In summary, the excellent study has limitations, which complicate the interpretation of the results. Addressing these limitations could strengthen and reinforce the statement of the study. The diagnostic and therapeutic management of AIE requires comprehensive imaging and CSF examinations, which must be repeated during follow-up to assess the treatment effect and long-term prognosis in AIE patients.

References

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