Autoimmune encephalitis after Head Trauma

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Introduction

Autoimmune encephalitis is an inflammation of the brain characterized by prominent neuropsychiatric symptoms that is associated with antibodies against neuronal cell-surface proteins, ion channels, or receptors[1]. Most common causes of autoimmune encephalitis include Anti-NMDA antibodies, paraneoplastic syndromes, and in rare cases, seroconversion from an HSV infection[2]. This report discusses a rare case of autoimmune encephalitis after a head trauma obtained from a ground level fall.

Case Description

A 55-year-old Hispanic male patient presented to the ED with worsening confusion. The patient started having intermittent episodes of confusion that began 2 months ago. He had difficulty recognizing his wife and children, visual hallucinations, illogical responses to questions, abrupt outbursts of anger and aggressive behavior towards family members. He also endorsed headaches, and episodes of blurry vision. On presentation to the ED, the patient was actively confused. His wife stated that his symptoms had progressed and his episode becoming more frequent. The patient denied fever, nausea, vomiting, chest pain, abdominal pain, loss of consciousness or seizures. Two months prior, the patient presented to an urgent care with a head laceration after a ground level fall at work. He presented with bleeding and swelling, no tingling or numbness, no reported loss of consciousness, nausea, vomiting, or altered vision. His wound was approximately 3 cm, superficial and 9 staples were applied.

On the physical exam, the patient was not oriented to place, time or situation. The pertinent positives from the neurological exam included dysmetria and agnosia. Patient remained afebrile and hemodynamically stable. Laboratory results showed a normal white blood cell count, cobalamin, folate, ammonia, T3, T4, and erythrocyte sedimentation rate. There was a negative blood culture, a negative toxicology screen, and a Streptococcus agalactiae positive urinalysis. A lumbar puncture was performed and pending cerebrospinal fluid analysis, the patient was started on Acyclovir for possible HSV Encephalitis and Ceftriaxone for an asymptomatic UTI.

An MRI of the head (Figure 1) revealed diffuse cortical edema and increased enhancement in the left cerebral hemisphere with prominence in the left temporal lobe and occipital lobes, Patchy cortical diffusion restriction was noted as well, more so, on the left occipital lobe revealing a diagnosis of encephalitis. Continuous EEG monitoring was initiated on the patient and Day 1 results showed five focal electrographic seizures emanating from the left Centro-temporal head region. Results of the CSF analysis showed high glucose: 154, high protein: 54, and normal white blood cell: 2. The CSF results were negative for bacterial, viral or fungal causes of encephalitis.

Based on radiologic and laboratory findings, autoimmune encephalitis was suspected, and pharmacologic intervention began appropriately. Acyclovir was discontinued owing to the negative HSV PCR. The patient was started on a 5-day course of methylprednisolone and IVIG plus Levetiracetam and Valproic acid for seizure activities.

On completion of his methylprednisolone and IVIG treatment, the patient showed significant improvement in functional, neurological, and cognitive status. He was oriented to person, place, and situation but required assistance with time. Repeat EEG showed absence of seizures and MRI showed total resolution of the previously noted enhancement within the left temporal and left occipital lobe. Patient was discharged on Valproic acid and a prednisone taper dose over twenty-one days

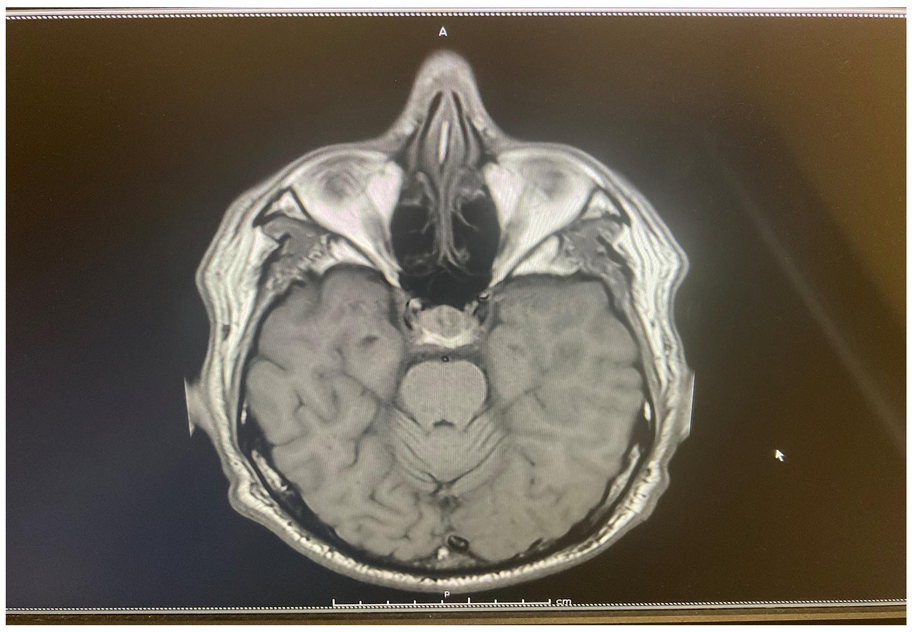

Figure 1: Magnetic resonance imaging of the Head on presentation Day 1

Conclusions

Autoimmune Encephalitis is largely a clinical diagnosis requiring 1) Subacute onset, altered mental status or psychiatric symptoms 2) New focal CNS findings or seizures 3) Reasonable exclusion of alternate causes like tumors, infections or metabolic syndromes.[3] In this case, metabolic causes of encephalitis were ruled out based on the patient's laboratory findings. In the absence of an obvious cause and clinical improvement upon treatment with methylprednisolone, it was safe to assume Autoimmune Encephalitis with an unknown Etiology. An argument could be made that this episode of encephalitis was likely triggered by the patient's history of a previous head trauma, but little is known on the relationship between trauma and autoimmune encephalitis and would be a possible area of research to explore.

References

1. Dalmau J, Graus F. Antibody-Mediated Encephalitis. *N Engl J Med*. 2018;378(9):840-851. doi:10.1056/NEJMra1708712
2. Leypoldt F, Titulaer MJ, Aguilar E, et al. Herpes simplex virus-1 encephalitis can trigger anti-NMDA receptor encephalitis: case report. *Neurology*. 2013;81(18):1637-1639. doi:10.1212/WNL.0b013e3182a9f531
3. Graus F, Titulaer MJ, Balu R, et al. A clinical approach to diagnosis of autoimmune encephalitis. *Lancet Neurol*. 2016;15(4):391-404. doi:10.1016/S1474-4422(15)00401-9

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