

INTRODUCTION

Autoimmune encephalitis is increasingly being acknowledged as a notable and common etiology of encephalopathy in the pediatric population. Despite the extensive array of antibodies identified for targeting the central nervous system, a considerable percentage of pediatric autoimmune encephalitis cases do not manifest detectable established antibodies, presenting a diagnostic dilemma. AIE encompasses validated syndromes predicated on clinical manifestations and autoantibody correlations. In contrast to adult AIE, the association with malignancy is less prevalent in the pediatric cohort.

AIMS / OBJECTIVES

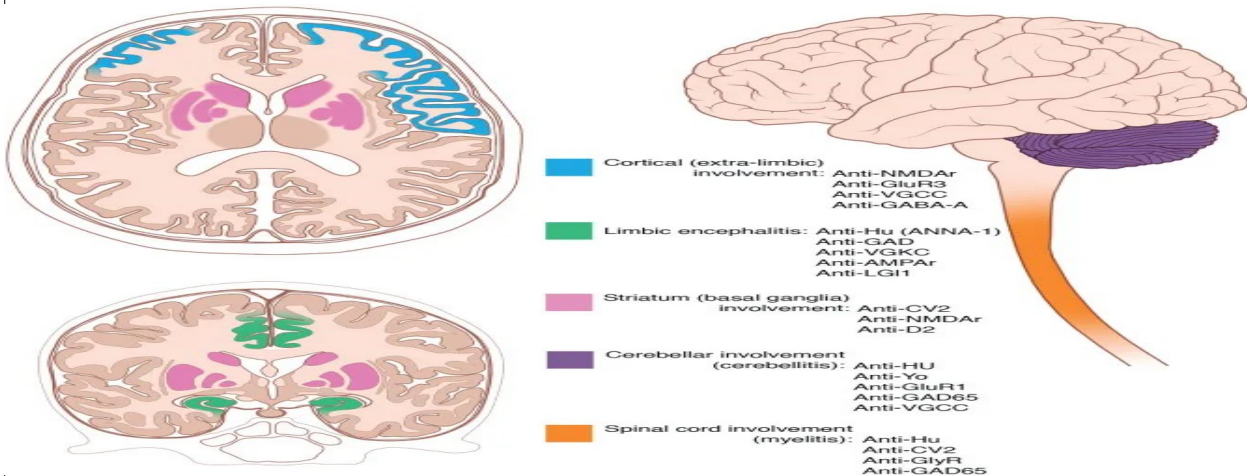
Early initiation of immunotherapy in autoimmune encephalitis has been found to improve patient outcomes.

MATERIALS / METHODS

We describe a case of a 11-year-old school going male student with encephalitis who presented to the emergency department with status epilepticus with no co-morbidities or seizures history. The patient had a fever 2 weeks prior lasting for 4days followed by 2 uneventful days before experiencing GTCS and focal twitches at school. Subsequently he developed Altered sensorium from 1 day and admitted in our Hospital. In ICU he developed right focal seizures followed by verbal abuse, spitting and involuntary movements of right upper and lower limb. Patient was started on IV antibiotics, anti-epileptics, neuroprotectives, IV Methylprednisolone and IVIG. Patient improved clinically after IVIG. Discharge MMSE Paediatric score was 32/37

RESULTS & DISCUSSION

CEMRI-hyper-intensity signal changes in sulcal spaces of bilateral cerebral hemispheres Acute Meningitis, CSF Analysis -2 cells lymphocytes, protein-41mg/dl glucose-67 mg/dl no pus cells negative for Gram staining, fungal, AFB staining, Anti-NMDA CSF antibodies negative, EEG-Epileptiform discharges. 72 hours holter report



CONCLUSION

Autoimmune encephalitis is increasingly being recognized in children. Anti-NMDAR encephalitis is the most common form. Children present with a polysymptomatic presentation including behavioral changes, psychosis, sleep disturbances, mutism, seizures, movement disorders, memory impairment, as well as other neurocognitive deficits. Diagnosis is based on suggestive history and ancillary investigations, including MRI, CSF analysis, and serology for autoantibodies. Treatment is based on immunomodulation of the acute episode followed by maintenance therapy, with earlier initiation being associated with better outcomes. Prognosis depends on the type of clinical syndrome.