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Case Report

Hyperventilation Induced Focal Seizures in an Adult - A Diagnostic Case Report

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Abstract

Introduction: Hyperventilation induced focal seizures in adults is a rare entity and it often gives a diagnostic clue in cases when most of the investigations are negative. We present a middle aged male with 5 days history of subacute onset headache, frequent anger outburst and irritability followed by seizures. His imaging with MRI Brain, CSF analysis and routine investigations gave no support to the suspected clinical diagnosis of acute encephalitis, but EEG showed typical temporo-occipital onset focal seizures induced by hyperventilation, which paved the way for us to suspect autoimmune etiology. Though serum and CSF autoimmune encephalitis panel was negative, in view of clinically suspected autoimmune encephalitis, a trial of steroids was given along with antiseizure medications.

Results: He was considered for slow taper of steroids along with azathioprine, with which he had significant improvement in his symptoms had no recurrence on 6 months of followup.

Conclusion: This case highlights the clinical significance of focal seizures induced by hyperventilation in adults and the etiological possibility to be considered particularly when autoimmune antibody panel is negative.

Keywords: Hyperventilation; Anti VGKC; Adults; EEG; Focal Seizure

Abbreviations

EEG: Encephalogram; CSF: Cerebrospinal Fluid; MRI: Magnetic Resonance Imaging; IgG: Immunoglobulin G

Introduction

About 10% of the new onset seizures in adults account to autoimmune etiology [1]. The phenomenon of hyperventilation induced focal seizures on electroencephalogram (EEG) is a rare entity among adults and contributes to less than 0.5% of the initial EEG assessments [2]. Recently, a number of cases have been reported to be specifically related to anti VGKC complex antibody mediated autoimmune encephalitis [3]. In a case of clinically suspected autoimmune encephalitis with negative yield from most of the investigations, induction of electrographic seizures by hyperventilation in appropriate clinical setting can

serve as an important diagnostic clue. Here we present one such patient, who had minimal clues from imaging, autoimmune panel and cerebrospinal fluid analysis, but had hyperventilation induced temporo-occipital seizures, who was successfully treated considering the possibility of seronegative autoimmune encephalitis.

Case Presentation

A middle aged male in his 40s had presented with 5 days history of new onset, mild to moderate hemicranial continuous headache, subtle behavioural changes in the form of frequent anger outburst and irritability with a single generalised seizure along with recurrent episodes of focal non motor seizures with preserved awareness characterised by visual aura, head and eye deviation towards right and right eye blinks lasting for few seconds to minutes. A clinical possibility of acute encephalitis and intracranial space occupying lesion localising to left occipital region was considered. His routine blood investigation including serum sodium was within normal limits. MRI Brain with contrast was essentially normal (Figure 1). CSF analysis showed elevated proteins (51 mg/dL) with normal cells and no oligoclonal bands. Serum and CSF autoimmune encephalitis panel including LGI 1 and CASPR 2 antibodies was negative. EEG showed no interictal epileptiform discharges but on induction of hyperventilation for 3 minutes, electro graphic seizure with clinical correlate originating from left temporo-occipital region lasting about 1-2 minutes was induced within 1.5 to 2.5 seconds on 3 separate occasions of EEG recording (Figure 2). Patient was treated with levetiracetam initially but continued to have frequent clinical seizures and had minimal response after addition of sodium valproate. Considering the clinico-radiological profile, the possibility of seronegative autoimmune encephalitis was considered [4]. He was started on methylprednisolone (1000 mg) for 5 days following which he had complete resolution of all his symptoms. He was then switched over to oral steroids (prednisolone 1gm/kg/day) along with addition of acetazolamide to his current anti seizure medications. On followup, he had no recurrence of seizures or other symptoms. Inspite of imaging and serum autoimmune panel being negative in our patient, hyperventilation induced temporo-occipital onset focal seizures in EEG formed a cornerstone to consider clinical autoimmune encephalitis and with immunotherapy, he had excellent clinical response, which further confirmed our diagnosis.

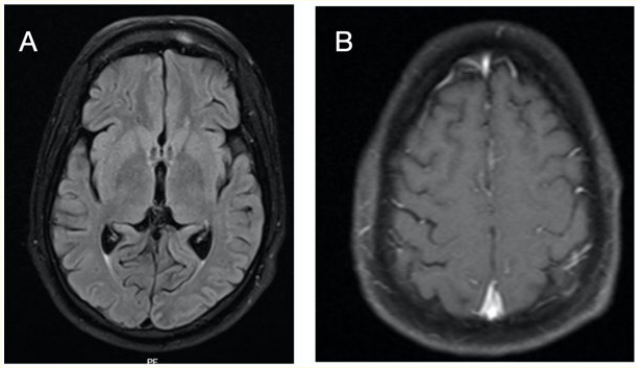

Figure 1: MRI (A) Axial T2 and (B) Axial T1 contrast showing no abnormality.

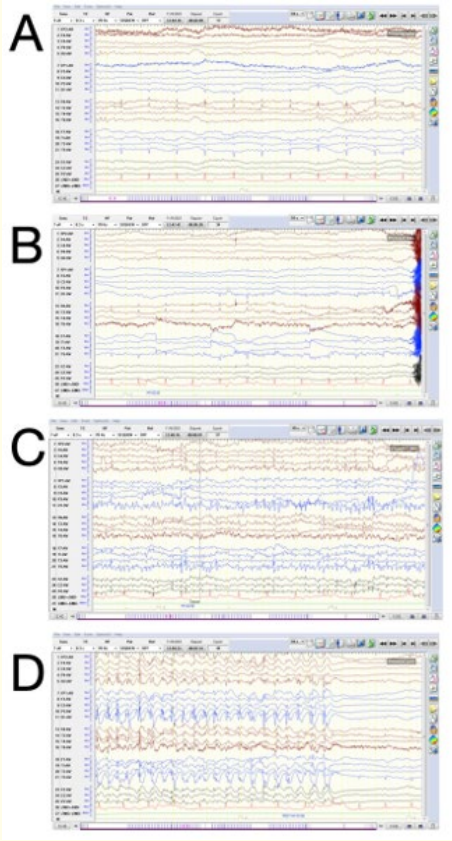

Figure 2: (A) EEG epoch showing normal record except for EKG artefacts. (B). At 2.20 minutes after the onset of hyperventilation, rhythmic beta activity is seen to evolve from left O1 and T5. (C) Better evolution over O1 and T5 leads with spread to adjacent leads (D). Post ictal slowing seen at about 1 minute post hyperventilation.

Discussion

Autoimmune encephalitis presents with impaired memory or cognition and seizures over days to weeks, which usually mimics infectious encephalitis. It can be broadly divided into several groups: pathogenic antibodies to cell surface proteins, antibodies to intracellular synaptic proteins, T cell disease with antibodies to intracellular antigens, those associated with other autoimmune disorders and paraneoplastic. The diagnostic criteria for autoantibody negative but probable autoimmune encephalitis was given by Graus., *et al*. as follows.

Fulfillment all 4 of the following criteria [4]:

1. Rapid progression (less than 3 months) of working memory deficits, altered mental status or psychiatric symptoms
2. Well - defined autoimmune syndromes to be excluded (e.g. limbic encephalitis or Bickerstaff encephalitis)
3. Absence of defined antibodies both in serum and CSF with any two of the following :
    - MRI abnormality suggestive of autoimmune encephalitis
    - CSF pleocytosis
    - CSF - specific oligoclonal bands or elevated CSF IgG index
    - Brain biopsy showing inflammatory infiltrates and excluding other disorders (e.g. tumour)
4. Reasonable exclusion of alternative causes

Voluntary hyperventilation as a means to provoke epileptic seizures was first introduced in 1924 and later, it has become a routine practice to perform this manoeuvre during EEG recording [10]. It commonly provokes epileptiform abnormalities in generalised epilepsies, particularly in children with absence seizures and about 11-25% of partial seizures [2]. Hyperventilation may have varied effect on EEG which includes: a) no effect b) clinical seizure with ictal EEG patterns c) Relative increase in epilpetifom discharges d) non epileptiform changes like slowing [2]. Hyperventilation for 5 minutes rather than 3 minutes increases the diagnostic yield by unmasking 16% of seizures and 30% of interictal abnormalities [5].

The mechanism by which hyperventilation induces seizures is obscure, but there are few proposed mechanisms. Hyperventilation causes hypocapnia and acute systemic alkalosis inducing tissue hypoxia at the cortical level causing cortical after discharge, chemically induced cortical spike foci and direct cortical response in addition to subcortical effect by causing cortical afferent locus shift from ascending reticular activating system to non specific thalamic system [6,7].

Hyperventilation induced focal seizures in adults gives an important clue on the etiology, especially anti-VGKC mediated encephalitis [9]. Seizures mediated by VGKC antibody encephalitis has excellent response to sodium channel blocking drugs and acetazolamide due to its pH mediated effects [8]. Acute management with steroids, plasma exchange and immunoglobulin therapy helps in the improvement of presenting symptoms. The immunomodulatory therapy must be decided based on the clinical response and individualised, however, the prognosis is good if identified early [10].

Recommendations

- Hyperventilation induced focal seizures in adults : Rule out autoimmune encephalitis especially anti-VGKC mediated
- Sodium channel blockers and acetazolamide are the effective anti seizure medications in VGKC mediated encephalitis
- With immunotherapy, the prognosis is usually excellent

Acknowledgements

None.

Conflict of Interest

None.

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