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An interesting case of acute psychosis with seizures

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Abstract: We present a case of an elderly female with acute onset of psychiatric symptoms in the form of auditory and visual hallucinations, disorientation and forgetfulness. She had two episodes of generalized tonic clonic seizures after admission to the hospital. Magnetic Resonance Imaging (MRI) of the brain showed bilateral hippocampal and para hippo campal hyper intensities suggestive of limbic encephalitis (LE). Infective and metabolic causes for the same were ruled out. Considering para neoplastic etiology, investigations for primary site of malignancy including Fluoro-deoxy glucose - Positron Emission Tomography (FDG-PET) were done which did not yield positive results. Autoimmune limbic encephalitis was the diagnosis after excluding other aetiologies and patient was successfully treated with immunosuppression with steroids. Limbic encephalitis is a medical disease which commonly presents to psychiatric department due to multiple psychiatric complaints. Early recognition of LE based on clinical findings and MRI study in absence of ant neuronal surface antibody test does improve the outcome and prognosis of the patient in long term.
Keywords: Hallucinations, autoimmune limbic encephalitis, fluoro-deoxyglucose- positron emission tomography (FDG-PET).

INTRODUCTION

The limbic system in the brain is the area where anatomy and medicine meet psychiatry [1]. It controls the cognitive and emotional processing, behaviour, motivation, spatial and long-term memory, and olfaction [2]. Limbic encephalitis (LE) is a rare disease, sub-acute in onset and characterized by short term memory loss, acute psychotic behavior in the form of agitation and irritability, delusions, hallucinations and seizures [3]. Diagnosis is often delayed because of abundance of psychiatric symptoms and paucity of specific neurological symptoms. LE can be multifactorial, with infective, metabolic, para neoplastic and autoimmune etiologies being the main causes. Diagnostic criteria and treatment protocols are also not definitive. We present a similar case with diagnostic dilemma.

CASE REPORT

A 68 year old diabetic female came with well-controlled diabetes and a history of sudden onset of recurrent malicious auditory and complex visual hallucinations, fearfulness, suspiciousness, disorientation and forgetfulness since 15 days. After admission to the hospital, she had two episodes of generalized tonic clonic seizures with post-ictal confusion and so she was transferred to the medical intensive care unit (ICU) for further management.

On examination in the ICU, her vital parameters, except blood pressure (190/100 mm of mercury), were stable. She was drowsy and disoriented to time, place and person. The plantar response was extensor bilaterally. There were no other focal neurological signs. Fundoscopy was normal. Following differential diagnoses were considered – hypertensive encephalopathy, metabolic encephalopathy and intracranial infections.

Complete haemogram, renal and liver function tests, serum electrolytes, urine examination, arterial blood gases, serum LDH (lactate dehydrogenase) and serum lipids were within normal limits. Blood sugar levels were well-controlled on oral antidiabetic drugs. Urinary and serum ketones were absent by dipstick method. Patient tested negative for Human Immunodeficiency Virus (HIV) and Australia antigen (HBsAg). Urine and blood cultures showed no growth. Electrocardiogram (ECG), chest X-ray and ultrasound of the abdomen-pelvis were normal. Magnetic Resonance Imaging (MRI) of the brain with contrast (Fig. 1) showed hyper intensities along the hippocampal and parahippocampal gyri bilaterally on T2 weighted and Fluid Attenuation Inversion Recovery (FLAIR) sequences. There was no restriction on diffusion and no enhancement on contrast. Radiological diagnosis was limbic encephalitis. A search for the cause of LE was undertaken.

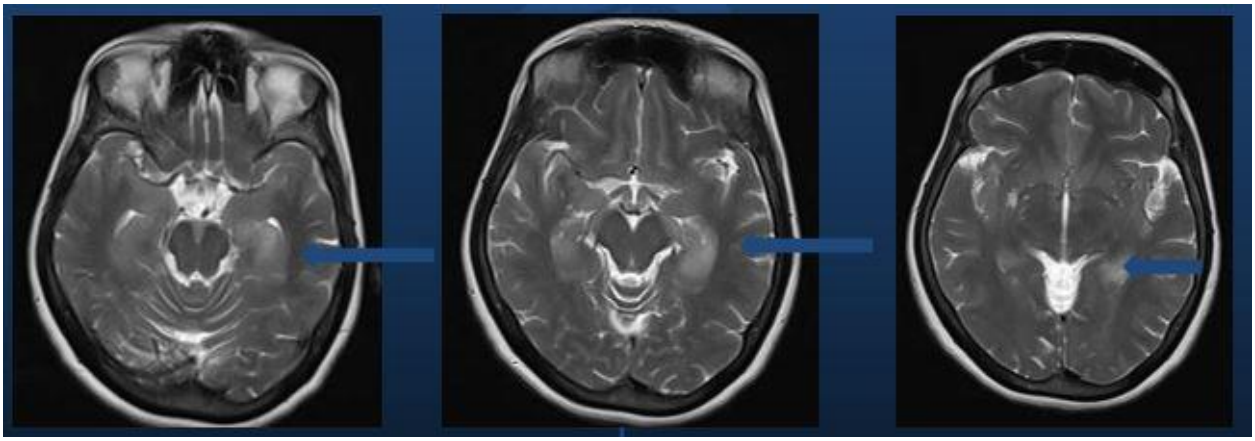

Fig-1: MRI Brain with Contrast: T2 and FLAIR sequences showing hyperintensities along the hippocampal and parahippocampal gyri without any diffusion restriction, suggestive of limbic encephalitis

Blood pressure fell within normal limits after putting the patient on tablet Telmisartan 40mg once a day and seizures were controlled with intravenous phenytoin sodium. Serum Ammonia, Vitamin B12 levels and thyroid functions were normal. Cerebrospinal fluid (CSF) examination showed clear fluid with 5 cells/cumm predominantly lymphocytes, sugar 65mg%, protein 44mg%. CSF culture showed no growth, CSF Adenosine Deaminase (ADA) and Polymerase Chain Reaction (PCR) assay for Herpes simplex (HSV) and Human Herpes virus-6 (HHV6) viral DNA were negative. Electroencephalogram (EEG) was normal. Thus, infectious and metabolic causes of limbic encephalitis were ruled out. The patient's general condition remained the same, i.e. she was drowsy with symptoms of acute psychosis. Considering para neoplastic etiology as a cause for LE, a search for primary neoplasm was undertaken. Contrast-enhanced Computerized Tomography (CECT) of the abdomen and pelvis, upper gastrointestinal endoscopy (with biopsy), echocardiogram, ultrasonography (USG) thyroid and mammogram were normal. CT thorax showed two old calcified lymph nodes measuring less than 1.5 cams in pretracheal region. Fluoro-deoxyglucose – Positron Emission Tomography (FDG-PET) scan showed focal increased FDG uptake in bilateral temporal lobes – parahippocampal region consistent with LE (Fig. 2) but no other FDG avid lesion anywhere on whole body survey to suggest occult primary malignancy.

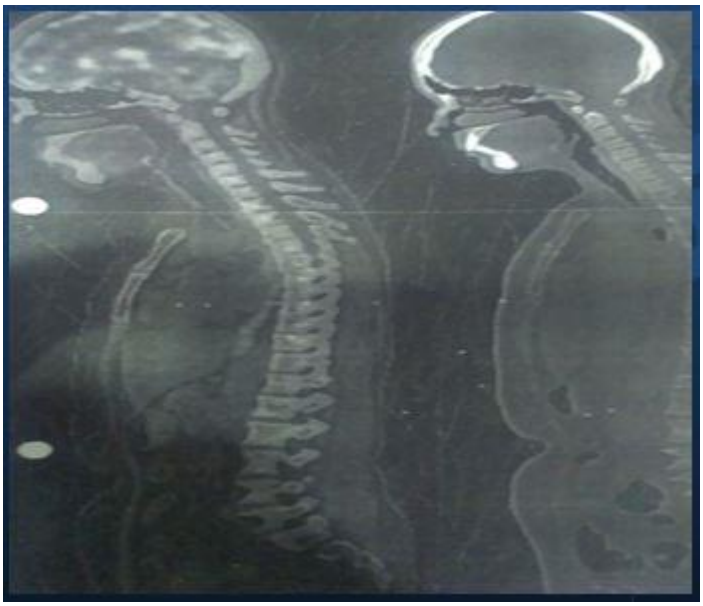

Fig-2: FDG-PET scan showing focal increased FDG uptake in bilateral temporal lobes – parahippocampal region consistent with LE; no other FDG avid lesion anywhere on whole body survey to suggest occult primary malignancy

Thus, there was no evidence of malignancy anywhere in the body. Anti-neutrophil Antibody blot analysis (ANA blot) and antimicrosomal antibody (thyroperoxidase) were negative. Antineuronal surface antibody (against N-methyl D-aspartate (NMDA) and

voltage gated potassium channels (VGKC)) screening could not be done due to financial constraints.

Patient was diagnosed as having non-paraneoplastic, noninfectious LE, probably autoimmune in origin and was given intravenous methyl prednisolone 1gm daily for 5 days followed by oral steroids in tapering doses. The patient's general condition improved and the psychiatric manifestations disappeared. She was discharged from the hospital with advice for regular follow-ups and is doing well till date.

DISCUSSION

Acute psychosis can be multifactorial. Diagnosing the cause of acute onset of progressive cognitive and behavioral problems including hallucinations can be very challenging. A case of acute onset of hallucinations and encephalopathy can be due to psychiatric disorders, metabolic derangements, infections, toxins and inflammatory mechanisms [4]. Some of these encephalopathies including limbic encephalitis can be caused by autoantibodies which are recognized by positive antibody markers and/or positive response to immunomodulatory treatment. The recent concept of aetiology of limbic encephalitis has expanded and it now includes various types of autoimmune limbic encephalitis associated with autoantibodies to intracellular or cell membrane antigens. Especially, new markers like antineuronal surface antibodies, antibody against voltage-gated potassium channels and N-methyl-D-aspartate, to name a few, have improved the diagnostic yield of LE greatly [5]. But, as mentioned in the article [6], in developing countries, due to economic constraints and non-availability, these assays are not done. Thus, we have to rely upon clinical and MRI findings, FDG-PET scanning, response to immunotherapy for finding out the cause of LE.

Thus, we successfully treated a case of acute psychosis and seizures which was diagnosed as nonparaneoplastic autoimmune LE. A high index of suspicion with relevant investigations in adults presenting with acute neuropsychiatric features can help us in diagnosing this rare, albeit increasingly recognized, form of autoimmune encephalitis. Typical MRI findings, negative FDG-PET scanning for occult neoplasia and clinical improvement with corticosteroids are helpful in diagnosis of autoimmune LE in absence of diagnostic but expensive antineuronal surface antibody screening especially in developing countries [6]. After the acute phase, patients are at risk of medically refractory seizures, chronic mood and anxiety disorders, readmissions to hospital and even loss of employment [7]. Thus, once diagnosed, a regular surveillance and follow-up is advisable.

CONCLUSION

To conclude, non-para neoplastic autoimmune limbic encephalitis is an important cause of rapidly progressive behavioral and cognitive dysfunction which is most often missed and unrecognized and which may lead to long term sequelae like refractory seizures, chronic behavioral changes and memory decline. MRI brain, FDG-PET scan, cerebrospinal fluid examination, electroencephalogram and serological testing for autoantibodies are useful aids in its diagnosis.

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