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A TYPICAL PRESENTATION OF VIRAL ENCEPHALITIS WITH PSYCHIATRIC MANIFESTATION AND QUICK; COMPLETE RECOVERY

Dhan Bahadur Shrestha,^{1*} Chiranjibi Panta,¹ Namrata Rawal,² Paawan Bhandari,³ Pradeep Manandhar,² Nagendra Katuwal²

¹Department of Medicine, Nepalese Army Institute of Health Sciences (NAIHS), Kathmandu, Nepal ²Department of psychiatry, Nepalese Army Institute of Health Sciences (NAIHS), Kathmandu, Nepal ³Department of Neurosurgery, Nepalese Army Institute of Health Sciences (NAIHS), Kathmandu, Nepal

*Correspondence to: Dr. Dhan Bahadur Shrestha, Department of Medicine, Nepalese Army Institute of Health Sciences (NAIHS), Kathmandu, Nepal. Email: medhan75@gmail.com

ABSTRACT

Viral Encephalitis is a common emergency condition, among the virus herpes simplex is on the top. Encephalitis usually present with clinical features of fever, headache, confusion with seizures but presentation can be atypical making the situation difficult and needs crucial judgement. Here we present an atypical case of viral encephalitis in a 32 years old male, initially presented with feature of depression with concomitant elicited history of conflict in the family confound-ing the situation. The case was managed successfully with multidisciplinary approach involving psychiatrist, neuro physician, and neurosurgeons in hospital for one month.

Key words: Atypical viral encephalitis, Insular cortex, Major depressive disorder, Midline shift

INTRODUCTION

Encephalitis is inflammation involving brain tissue, is strictly diagnosis of pathologist. Its incidence in west ranges from 0.7 to 13.8/ 100,000 and common in children than adults¹. Encephalitis caused by Herpes simplex virus (HSV) is the commonest viral encephalitis and is usually due to HSV-1 (10% due to HSV-2). Other less common agents are Cytomegalovirus (CMV) and Varicella zoster virus (VZV); common in immunocompromised¹.

Viral encephalitis typically presents with headache, fever, confusion with convulsions and focal neurological symptoms in some cases but patient may not have such typical features in all. A good history (from the relatives of comatose patient) and thorough general examination, is crucial in probing the probable cause of encephalitis^{2,3}. In acute encephalitis, magnetic resonance imaging (MRI) play diagnostic role, in Herpes Simplex Encephalitis (HSE) area with focal edema over temporal cortex, orbital cortex of the frontal lobes, insular cortex and angular gyrus is common radiological findings².

Management is mostly supportive but if antiviral therapy is not instituted to patients with herpes encephalitis the mortality is high².

CASE HISTORY

A thirty-two years old male serving soldier referred from military field hospital, Surkhet, Nepal; brought to our Hospital, on 7th April 2016 with presenting complaints of poor social interaction, self-care and abnormal behavior for last four days. There was no associated fever, headache and vomiting. No history of psychological/neurological disorders other than conflict with spouse for 3-4 month could be elicited.

Examination revealed depressed look with slow mentation and poor attention, concentration, insight and judgement. So, he was initially admitted and treated on the line of major depressive disorder with Lorazepam 2mg twice daily(BD), Escitalopram 10 mg and Omeprazole 20 mg but next day evening detected to have high grade fever of 1030F, decreased level of consciousness (Glasgow coma scale (GCS)=9/15). Patient was toxic looking with

decreased O2 saturation (SPO2=50%), fall in blood pressure (90/50 mmHg) so, shifted to intensive care unit (ICU).

Urgent CT head ordered and showed diffuse cerebral edema with mass effect, hypodense mass lesion on left frontoparietal region. Immediately supportive management started on the line of encephalitis; antiviral not given due to late diagnosis. Later, patient was noted of absent movement of right upper limb. Contrast enhanced computed tomography (CECT), showed left insular cortex enhancement and Contrast MRI showed high-intense signals in the temporal cortex and hippocampus on Fluid Attenuated Inversion Recovery (FLAIR) technique, suggestive of encephalitis.

Fifth day post admission onwards patient started to improve with spontaneous eye opening and obeyed simple commands and stable vital parameters. In between there was behavioral problem, after gaining some insight and improved behavioral abnormality patient was discharged after 26 days of hospital stay asking for follow up after one month keeping him under tab atorvastatin 10 mg nocte, tab olanzapine 5 mg BD and tab pantoprazole 40 mg BD. He was followed up 1 month after discharge from hospital and found to have dramatic improvement. There were no residual symptoms and sign and/or residual cortical deficit of the previous encephalitis. He is under regular follow up, last follow up was after a year of the event.



A. CECT image showing enhancement over insular cortex and diffuse edema and midline shift towards right and subdural hematoma; B, C. T_2 weighted MRI scan showing frontotemporal enhancement



D. T₁ weighted FLAIR film; E. Contrast enhanced MRI. F. CECT during follow up 1 month after discharge

DISCUSSION

Encephalitis denotes inflammation of brain tissue mostly viral in origin and common in immunocompromised individuals. It should be suspected in a patient having febrile illness having headache, altered consciousness and features of cerebral dysfunction. Not all the patient present with classical pictures but patient usually

present in the hospital with four group of symptoms due to cerebral dysfunction and includes: deranged cognition (speech, memory and orientation abnormalities, etc.), behavioral problems (change in personality, disorientation, agitation, psychosis), focal neurological disturbances (hemiparesis, dysphasia, anomia), and convulsions⁴.

In atypical presentation like in this case radio-imaging may help diagnosing disease. HSE in imaging will be characteristic low attenuation, mass over temporal area and insular cortex, enhanced lesion and hemorrhage in late period of illness but in early stage it could be normal. Typical initial findings include edema of gyri in T_1 weighted imaging imaging(WI) and high signal intensity over the cingulate gyrus or temporal cortex on T_2 imaging, diffusion weighted imaging (DWI) and FLAIR and hemorrhage in later period^{5,6,7}.

In this case, we have suspected encephalitis due to deteriorating clinical condition and typical radiological finding suggestive of Herpes simplex encephalitis with involvement of insular cortex and hippocampus with associated subdural hemorrhage. Though the clinical scenario was puzzling due to depressive features and behavior abnormalities with no features suggestive of meningitis or systemic infection; but fever and alteration of vital parameters following admission has changed the scenario. Though encephalitis suspected lumbar puncture was not done due to edema with midline shift but managed with supportive measures using osmotic diuretics and steroid.

In literature though there is reported cases of atypical viral encephalitis in immunocompromised individuals and having the poor prognosis8 but no such atypical presentation in young immunocompetent individual is reported. Usually mild/atypical HSE develops among immunocompetent individuals and is typified by the absence of focal lesions and gradual deterioration, if antiviral therapy is not given and only detected by screening the cerebro-spinal fluid (CSF) specimen.⁹

To conclude viral encephalitis though less common can occur in immunocompetent individual with atypical presentation; can be grave if not managed crucially and if general supportive measure is provided well there can be dramatic improvement preventing the dire consequences. Radio imaging is vital for the diagnosis and clinician should be highly suspicious of encephalitis if individual present with features of cerebral dysfunction though there can be absence of systemic features of infection or meningitis.

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