Case Report

VIRAL ENCEPHALITIS PRESENTING AS CVA (STROKE)

AM.P.Gorkhaly*, A. Poudel**, Rewati Raman Malla***

Abstract

Acute viral encephalitis is due to direct invasion of the brain by virus with herpes simplex type 1 being most common. Here is a case being presented and treated as ischemic stroke, which after deterioration at the first Hospital, was diagnosed as Herpes Encephalitis at our Hospital. Despite the fact that, patient was critical and ICU care was given for more than 2 weeks and total hospital stay was around 2 months, after being treated with injection Acyclovir and steroids, patient recovered progressively. Now the patient is neurologically normal and is performing his day to day activities. Thus, there is need to evaluate for secondary causes when patient with stroke deteriorates despite of usual management.

Keywords

Acyclovir, CT scan, Herpes Encephalitis, Ischemic stroke.

Introduction

Acute viral encephalitis is due to direct invasion of the brain by virus, and the signs and symptoms result from this invasion and from the inflammatory change which it indices in the brain parenchymaIn Europe, the most common cause of viral encephalitis is herpes Simple, which probably reaches the brain via the olfactory nerve. An encephalitis is the most serious type of disease produced by HSV in the normal immunocompetent host

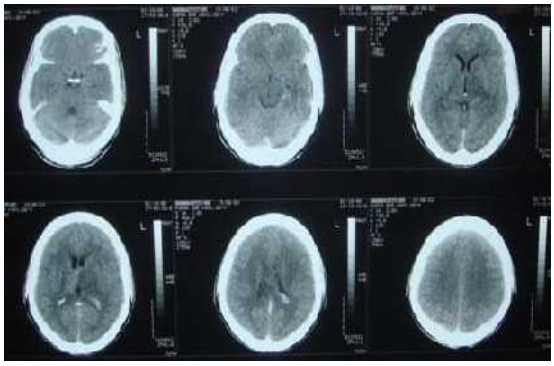
Here, we are reporting a case of Herpes encephalitis presented with right sided hemiplegia, fever, headache and altered level of consciousness which was diagnosed and managed in the line of ischemic stroke and later deteriorated. Encephalitis presenting with hemiplegia often leads to a clinical dilemma in our setting. Considering HSE as an alternate diagnosis in the early stage of the illness supported by the imaging and the clinical presentation, supportive and appropriate treatment leads to the complete recovery. And this case reporting is to make the practicing physicians and neurologists aware about the cause of Herpes Simplex Virus Encephalitis presenting as hemi paresis mimicking CVA.

Case reporting

The patient was referred from another tertiary level hospital of Kathmandu, where

*Associate Prof., Dept. of Medicine, Bir Hospital, NAMS
**Dept. of Medicine, Bir Hospital, NAMS
***Dept. of Medicine, Bir Hospital, NAMS

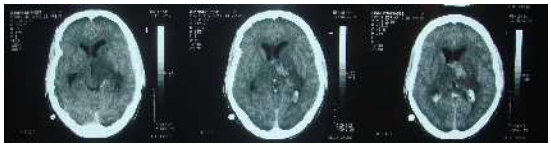
he received primary treatment and was managed conservatively with aspirin tab for ischemic stroke (CT Scan no. 1).



(CT scan no.1, on 1st Oct., 2006 showing left basal ganglia region infarction)

His general condition deteriorated rapidly for which he was referred for the ICU support and the needful. In our hospital he was admitted on 2nd October 2006 and was discharged on 26th November 2006.

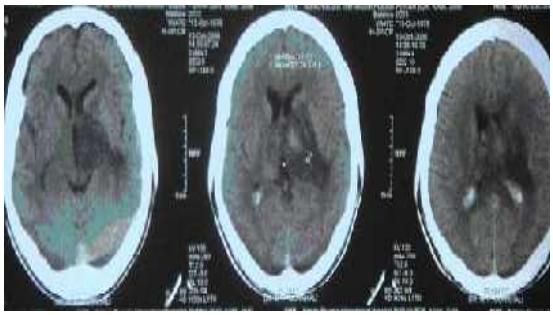
We received a 30 years old male, cook by profession returning after a high altitude trip above 5000 meters. He was having chief complaints of headache and fever for 7 days, unconsciousness for 2 days, and few episodes of vomiting with right sided hemiplegia and aphasia. All these began when patient was apparently not having any complaints before the trip and started after 2 days of return to Kathmandu. This all started with sudden headache followed by fever which progressed over 2 days to altered level of consciousness and weakness of the right side of body, for which he was taken to the hospital. During the stay at hospital he deteriorated further. There was no suggestive history for HTN, DM or past similar episodes. He refuses any contact with similar patients. He has no allergies. He was not on any regular medications. He is neither alcoholic nor a smoker. He'd never been admitted at the hospital for any reason and does not give any significant surgical history. At the time of admission pulse was 72bpm, BP of 120/70 mm of Hg. He was afebrile and acyanotic. He was having SPO2 of 96% at room air. Chest showed bilateral crepitations. GCS was E4M4V6, neck rigidity and kerning's sign positive, with down going planters. CSF report showed total count of 50 cells/hpf, with all polymorph nuclear cells, protein 54gm/dl and sugar 54gm/dl, with clear consistency and no RBCs nor xanthochromia . The follow up report for which showed no bacterial growth. LP was repeated at our center, and CSF report was suggestive of viral meningitis. Since, the patient was already started on iv Ceftriaxone 2 gm 12 hrly, mannitol and steroids but his general condition were rapidly deteriorating, we reevaluated for the possible reasons. CT head was ordered which showed hemorrhagic foci on left basal ganglia region (CT scan no.2).



(CT scan no.2, on 4 Oct., 2006 showing acute hemorrhagic infarct in the left basal ganglia region)

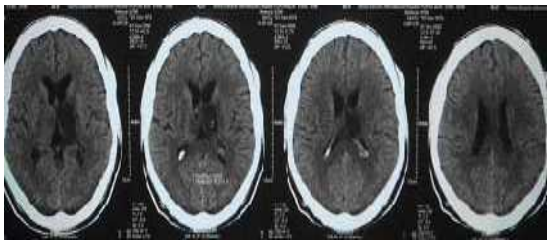
This lead to suspicion of Herpes Simplex Virus Encephalitis. Taking the CSF for immunologic testing, we promptly started the

patient on Acyclovir 10mg/kg and steroids. The CSF was positive for IgM for Herpes Simplex Virus 1. Acyclovir was continued for total of 21 days. The CT scan repeated showed Acute infarct involving left thalamus, internal capsule and basal ganglia with focal hemorrhagic transformation, along with small focal infarction of right thalamus(CT scan 3).



(CT scan no.3, on 13th Oct.,2006 showing extension of the hemorrhagic foci and right thalamus infarct)

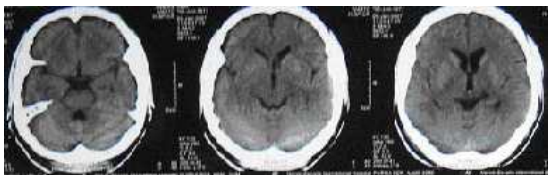
Through out the course of his hospital stay patient gradually regained considerable amount of motor, sensory, cranial nerve functions. During the 15 days stay at ICU patient had proximal limb power of 3+/5 and distal limb of 2/5. During the stay at the ward for 1 month he had proximal limb power of 4+/5 and distal limb was 3/5. During his hospital stay, we re evaluated with the repeated CT scan which showed progressive improvement in the size of lesion (CT Scan 4).



(CT scan no. 4, on st Nov., 2006. showing resolving hematoma with left basal ganglia infarct)

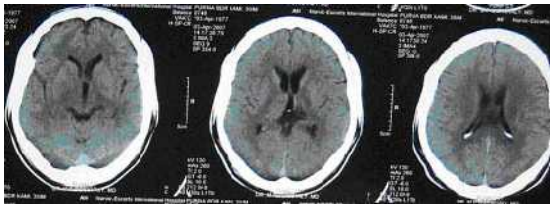
(Photo no. 1 showing his neurological status On 2nd Nov., 2006

With this significant improvement and good activities of daily living which he was able to do, he was discharged to follow up with reports of Herpes Simplex Virus IgG Antibody serology test. At the time of discharge patient was aphasic and could walk but could not stand from squatting position. His CT done at discharge showed normal scanning of the brain (CT Scan 5)



(CT scan no.5, on 5th Jan., 2007 showing normal scan)

Even though the repeat CT scan showed infarction of the left basal ganglia region (CT scan 6) at a later visit, he had regained significant amount of CNS functions. He was able to talk with good fluency, was able to walk normally and normal higher mental function. He had recovered completely from the right sided hemiplegia and was leading a normal life.



(CT scan no.6, on 3rd April, 2007, showing non hemorrhagic infarction of left basal ganglia)

(Photo no. 2, on 3rd April with patient living happily with the family with neurological recovery)

Discussion

HSV 1 is responsible for most of the non genital 'above the belt' infection. HSE has become the most common form of sporadic and often fatal or disabling encephalitis worldwide. With HSE has an estimated prevalence of 0.1-0.4 per 100,000 populations per year.. With incidence of 2 to 3 per million of population. This is almost certainly an underestimate for it seems likely that milder forms of the disease go unreported and perhaps unreconized[1]. Changes of central nervous system are results of primary infection or activation of latent HSV 1, HSV 2.[1] An important feature of HSV1 encephalitis is its proclivity to involve the temporal lobes and orbital frontal areas, where it produces hemorrhagic, necrotizing encephalitis.. The mechanism by which the virus reactivates and penetrates into the CNS is still not fully understood. A timely diagnosis is crucial so that early treatment can be established within the first four days of the infectious process. By so doing, it becomes possible to raise the chances of survival by over 50%.[2].

The clinical presentations of HSE are protean. The onset may be explosive but is more often insidious. Prodromal symptoms commonly last between 4 and 10 days and consist of malaise, fever, headache, and irritability. In more severe cases there is meningial irritation, depression of conscious level, and seizures which may be generalized or focal. Signs of frontal and temporal lobe dysfunction follow, with personality changes, memory loss, and psychiatric syndrome with hallucinations and – as the disease progress-hemi paresis and parietal syndromes. In a healthy individuals HSV encephalitis is provoked by certain febrile illness(e.g. Common cold, Pneumonia); direct sunlight; stress; trauma; menstruation; immunocompromised. Considering the clinical features, exposure to

the high altitude leading to direct sunlight and stress as a contributing factor to provoke the HSE in our patient. There may be unreported or un-noticed episodes of other herpes simplex infection. So, the index of suspicion of HSE should always be high for a patient presentation with the typical features of encephalitis such as fever, headache, confusion, and clouding of consciousness[3]. It is essential to commence early treatment with intravenous acyclovir in patients suspected of having HSE because of the remarkable safety and efficacy of this drug and the dangers of delaying potentially effective treatment of life threatening disease.[4] As we can't predict the outcome of patients with HSE in the early stage of illness and delay of treatment may case disaster, early diagnosis and prompt acyclovir initiation are important requirements for successful managemtnt.[5]

The diagnosis of HSE is usually established from the combination of the clinical and investigative features.[6] CT, MRI and SPECT are useful tools in early recognition of herpes encephalitis. The application of PCR is prompt and specific diagnosis of herpes simplex virus infections of the brain..The use of polymerase chain reaction (PCR) techniques to amplify the genome of herpes simplex virus (HSV) from cerebrospinal fluid (CSF) has become the diagnostic procedure of choice. However several problems for the PCR in HSVE remain, as follows: the discrepancy in results based on differences of minimum detection sensitivities, single PCR and nested PCR and clinical pseudo negative results which depends on the day of CSF sample collection after onset .[5] Along with these limiting factors and the cost, we couldn't send the CSF for the PCR and taken as a limiting factor at the initial stage of treatment.

The CT scan no 6 showed non hemorrhagic infarct over left basal ganglia, though patient was neurologically normal and was leading normal life. There is one case reporting with an intracranial hematoma after successful and complete recovery with acyclovir therapy. Location of the hematoma corresponded to the initial encephalitis area involving the medial temporal lobe structures. Despite this late neurologic complication, after 18 days of symptoms onset, the patient had a favorable neurological outcome[8]. This is 2nd case reported and to the best of our knowledge, ours is the 3rd case reported of the unusual, rare and neuro imaging complication of acute ischemia over basal ganglia region after complete recovery from treated HSVE with favorable clinical outcome. During the course of illness, in addition to other features, patient is found to be aphasic noted after gaining consciousness. Aphasia was improving along with good neurological outcome and now the patient can talk and communicate normally. Similar case has been reported as Herpes Encephalitis presenting as mild aphasia.[9].

Regarding Anti herpes virus drug, in both Japanese and IMHF guidelines acyclovir is consistent with the first choice, and it is recommended that its administration would be started as soon as HSE is suspected on the basis of clinical picture, CT, MRI, EEG, or CSF findings. A recent Japanese study shows the

efficacy of a combination therapy of acyclovir and corticosteroid for this disease[10]. A poor outcome was evident with older age, lower Glasgow Coma Scale scores, and if corticosteroid isn't administered along with acyclovir. Combination therapy employing both acyclovir and corticosteroid is thus suggested to be useful for achieving a better outcome[11].

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