Case Report

Imaging spectrum of viral encephalitis: A case series

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Abstract

Encephalitis refers to an acute, diffuse inflammatory process affecting the brain and viral encephalitis being the most important cause. Imaging of patients with viral encephalitis reveals edema in the affected brain parenchyma with possible diffusion restriction in the acute phase probably due to cytotoxic edema. Depending on the virus grey and white matter of the brain may be involved or a distinct site may be involved such as temporal lobes in HSV encephalitis. Viral encephalitis has a predilection for young and elderly. HSV encephalitis is the most commonly diagnosed viral encephalitis in developing countries. ADEM is an important differential diagnosis to be considered which tends to involve the white matter and the distribution is usually asymmetric. In the present case series we have presented HIV, Herpes, Rota virus, and Dengue manifestations of viral encephalitis. The purpose of this montage is to ensure that the clinician includes the specific virus in the differential diagnosis.

KEY WORDS: Encephalitis, HIV, Herpes, Rota virus, Dengue, imaging

Introduction

Most common clinical manifestations of viral encephalitis include disorientation, speech disturbances and behavioral changes with one-third of the patients developing seizures.[1] With the advent of CSF PCR, more subtle manifestations of viral encephalitis can be recognized. Although most viral encephalitis present acutely, subacute, and chronic presentations can be caused by CMV, VZV, and HSV, especially in patients immunocompromised as a result of HIV or immunosuppressive drugs.[2] It is important to distinguish ADEM from acute infectiousencephalitis, acute non-infectious encephalitis, and an acute metabolic or toxic encephalopathy.[3] Acute encephalitis constitutes a medical emergency. Early diagnosis is important for appropriate management. MRI imaging of brain is the investigation of choice and the diagnosis has to be confirmed by PCR for the virus in the CSF. CT scan is usually warranted before attempting lumbar puncture to look for signs of raised intracranial tension.

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Case 1: A 41-year-old gentleman who was detected to be HRV positive 3 months back with complaints of tiredness since 1 month, personality changes, confusion, memory loss, weakness of bilateral upper and lower extremities, and choreoathetoid movements of left hand with fine tremors. Imaging features were suggestive of HIV encephalitis (Figure 1).

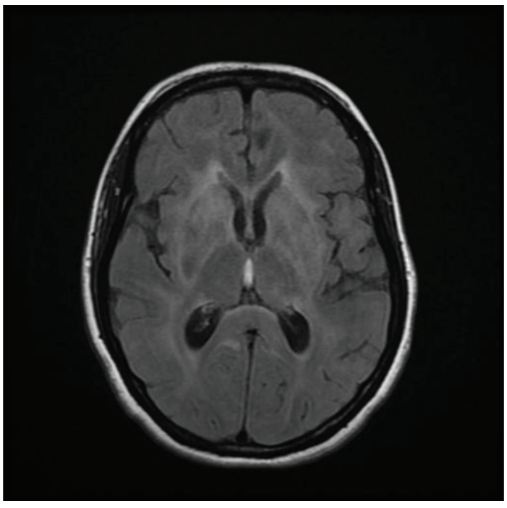

Figure 1: Axial T2 FLAIR image showing hyperintensities in bilateral basal ganglia, periventricular white matter and subcortical regions.

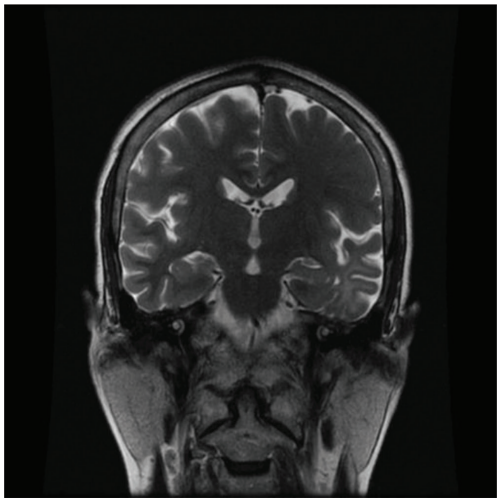

Figure 2: Coronal T2W–Fast spin echo (FSE) image showing hyperintensity in the deep white matter of left temporal lobe with cortical thickening suggestive of Herpes encephalitis.

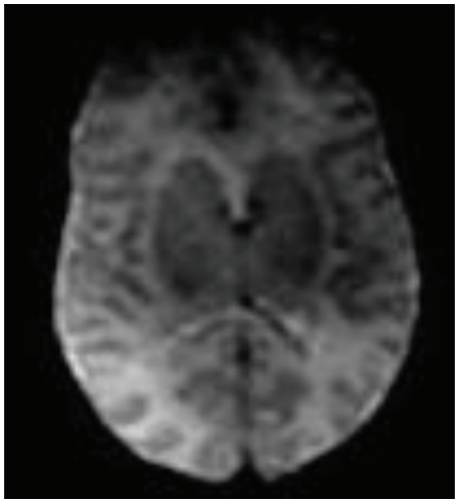

Figure 4: Axial T2W image showing hyperintensities in deep white matter of bilateral fronto-temporo-parieto-occipital lobes and corpus callosum.

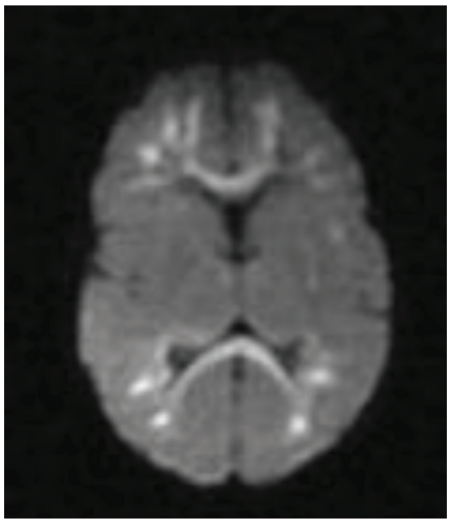

Figure 3: Axial DWI image showing diffusion restriction in deep white matter of bilateral fronto-temporo-parieto-occipital lobes and corpus callosum with sparing of subcortical U fibers.

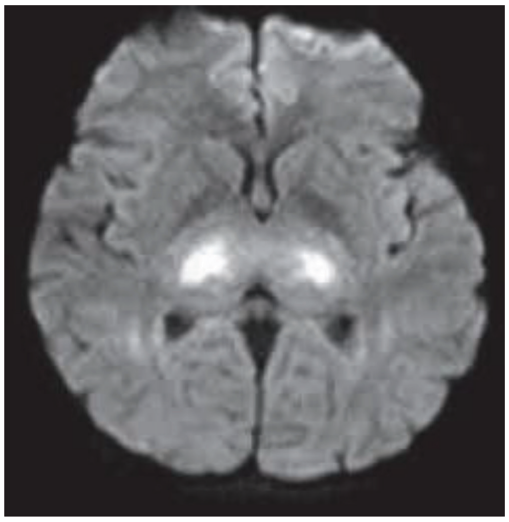

Figure 5: Axial DWI image showing diffusion restriction in bilateral thalami.

Case 2: A 56-year-old lady with lethargy and 15 kg weight loss over the past year was admitted to the hospital with fever, altered mental status, altered mental status, and left hemiparesis. CSF revealed lymphocytic pleocytosis, elevated protein and glucose and positivity for IgM antibodies against HSV. EEG showed focal slowing and sharp waves. Imaging showed T2 hyperintensity in left temporo-parieto-occipital lobes with cortical thickening. Features were suggestive of Herpes encephalitis (Figure 2).

Case 3: A 5-month-old previously healthy male child was admitted to the hospital with a 2 day history of nonbloody diarrhea and vomiting. On the day of admission he developed fever and had a generalized tonic-clonic seizure lasting 2 min. On arrival to the hospital, he was dehydrated and drowsy. CSF analysis showed lymphocyte count of 2.4×10^9/liter and bacterial cultures of blood and CSF were negative. Imaging features were suggestive of Rotavirus encephalitis. Clinically the child improved over the first 24 hours and subsequently made full recovery (Figures 3 and 4).

Case 4: A 22-year-old pregnant woman at 34 weeks of gestation with viral fever, erythema multiforme and myocarditis who presented with complaints of acute onset weakness in right lower limb and hyper-reflexia. Laboratory investigations showed thrombocytopenia and dengue positive serology.

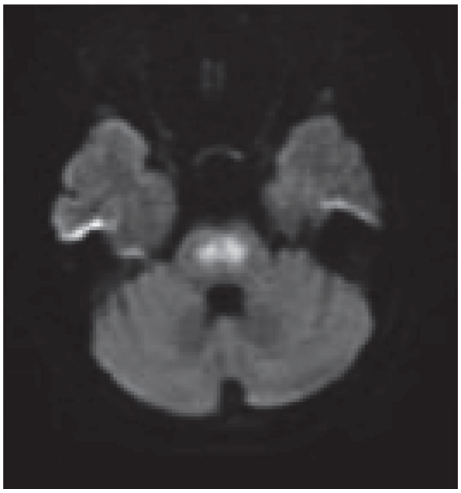

Figure 6: Axial DWI image showing restricted diffusion in pons.

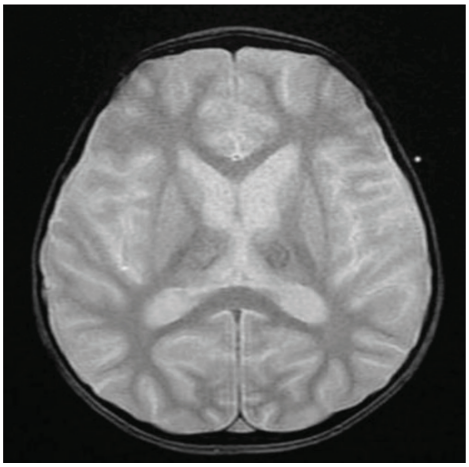

Figure 7: Axial gradient recalled echo image showing blooming bilateral thalami.

Imaging suggested hyperintensity in bilateral thalami, pons and bilateral perirol and ic post-central gyri. DWI images showed diffusion restriction in bilateral thalami and pons. Blooming was noted on GRE in bilateral perirol and ic post central gyri. Features were suggestive of Dengue encephalitis. The diagnosis of dengue was confirmed by positive serology for Ig Mantibodies and NS-1 antigen positivity (Figure 5–7).

Discussion

HIV causes a type of subacute encephalitis, it is important in so far as its associated immunosuppression predisposes the individual to viral encephalitis caused by, for example, HSV-1, HSV-2, VZV, and cytomegalovirus (CMV). HIV crosses the intact blood-brain barrier, and the virus has been cultured from the brain, nerve, and cerebrospinal fluid of patients at all stages of disease.[4,5] The imaging findings of AIDS dementia complex are frequently referred to as HIV encephalitis. On magnetic resonance (MR) images, a diffuse cerebral atrophy with symmetric, patchy or confluent areas of T1 and T2 prolongation are seen within the periventricular and deep white matter of patients with AIDS dementia complex. Often, there is a frontal predominance that may include involvement of the genu of the corpus callosum.

HSE can be the result of a primary infection, a reactivation of latent HSV, or a re-infection by a second HSV.[6] About 90% of cases of cases of HSE are caused by HSV-1, with 10% due to HSV-2, the latter usually being the cause of HSE in immuno compromised individuals. Herpes encephalitis is the most common cause of sporadic viral encephalitis with a predilection for temporal lobes. HSV encephalitis is associated with high mortality and in adults typically causes asymmetric bilateral FLAIR/T2WI hyperintensity in the medial temporal lobes, insular cortex and posterior–inferior frontal lobes with relative sparing of basal ganglia and neonates in which it causes a disseminated infection.

Rotavirus is the primary cause of severe gastroenteritis in children in the winter and spring. The incidence of CNS involvement at rotavirus gastroenteritis as 25.3%.Concomination of CNS involvement to rotavirus infection can represent by different clinical findings. Meningitis, encephalitis, encephalopathy, febrile and afebrile convulsions, hemorrhagic shock, Guillian Barre syndrome and Reye syndrome are the reported neurological entites until now.[7] Ushijima et al[8] established rotavirus both at intestinal and CSF observation, so by this knowledge rotavirus seem to make CNS invasion after a primary intestinal infection. Electrolyte imbalances, destructıon of bood brain barrier by fever or encephalopaty and encephalitis are the causes for occurence of convulsions.[7]

Dengue viral infections are very common in Southeast Asia and all 4 serotypes are found. It is known to cause dengue fever and dengue hemorrhagic fever. Encephalitis has been well reported and is thought to occur with severe dengue infection leading to liver failure, shock, coagulopathy and leading to cerebral insult. Dengue encephalitis patients usually present with fever, altered sensorium, thrombocytopenia and high antibody titers at the time of admission. Encephalitis is a very common neurological complication with dengue fever and is due to direct neuronal infiltration by the virus. Dengue encephalitis is a well-recognized and common entity with incidence ranging from 0.5% to 6.2%.[9] It may be due to intracranial bleeding due to thrombocytopenia, cerebral hypoperfusion or cerebral edema.[10] Dengue virus and Ig Mantibody in the CSF has been reported in patients with dengue encephalitis.

Conclusion

In acute viral encephalitis, findings include white matter signal intensity changes, cerebral edema which may progress in later stages to infarction, hemorrhage and brain atrophy. MRI features of viral encephalitis are highly variable according

to the offending virus and patient age. Imaging changes must always be evaluated in conjunction with the clinical symptoms,signs, and laboratory abnormalities, particularly the presence of a CSF pleocytosis. Unlike bacterial and fungal meningitis in which imaging abnormalities are not specific for a particular agent, many virus infections of the CNS produce MRI abnormalities not seen by any other infection.

References

1. Raschilas F, Wolff M, Delatour F, Chaffaut C, De Broucker T, Chevret S, et al. Outcome of and prognostic factors for herpes simple xencephalitisin adult patients: Results of a multicenter study. Clin Infect Dis 2002;35:254–60.
2. Fodor PA,Levin MJ,WeinbergA, Sandberg E, Sylman J, Tyler KL. Atypical herpes simplex virus encephalitis diagnosed by PCR amplification of viral DNA from CSF. Neurology 1998;51:554–9.
3. Coyle PK. Postinfectious encephalomyelitis.In: *Infectious Diseases of the Nervous System*, 1st edn. Davis LE, Kennedy PGE (Eds.).Butterworth-Heinemann, 2000. pp. 83–108.
4. Chiodi F, Keys B, Albert J, Hagberg L, Lundeberg J, Uhlén M, et al. Human immunodeficiency virus type 1 is present in the cerebrospinal fluid of a majority of infected individuals. J ClinMicrobiol 1992;30(7):1768–71.
5. Garcia F, Plana M, Vidal C, Cruceta A, O'Brien WA, Panteleo G, Pumarola T, et al. Dynamics of viral load rebound and immunological changes after stopping effective antiretroviral therapy. AIDS 1999;13(11):F79–86.
6. Tyler KL. Herpes simplex virus infections of the central nervous system: Encephalitis and meningitis, including Mollaret's. Herpes2004;(Suppl 2):57A–64A. [Medline].
7. Abe T, Kobayashi M, Araki K, Kodama H, Fujita Y, Shinozaki T, et al. Infantile convulsions with mild gastroenteritis. Brain Dev 2000;22:301–6. [Pub Med: 10891637]
8. Ushijima H, Tajima T, Tagaya T. Rotavirus and the central nervous system. Brain Dev 1984;6:215.
9. Misra UK, Kalita J, Syam UK, Dhole TN. *Neurological manifestations of dengue virus infection. J Neurol Sci 2006;244:117–22.*
10. Varatharaj A. Encephalitis in the clinical spectrum of dengue infection. Neurol India. 2010;58:585–91.

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