Original Research Article

Magnetic resonance imaging findings in viral encephalitis - A pictorial essay

Kalaivani J[1], Rajeswaran R[2*], Prashanth J[3]

[1,3]Consultant Radiologist, [2]Professor, Department of Radiology, Sri Ramachandra Institute of Higher Education and Research No.1 , Ramachandra nagar, Porur, Chennai, Tamil Nadu 600116, INDIA.
Email: vanijayaraman58.om@gmail.com

Abstract

Viral encephalitis by definition is the result of human virus affecting the brain and sparing the meninges. Encephalitis can involve any age group from children to old people. The severity of the disease depends on the viral agent and the host immune system. When the infection involves the meninges besides brain it leads to meningo -encephalitis. The patient usually presents with flue like symptoms: fever, sore throat, cough, headache and malaise. Some patients also present with altered sensorium when associated with severe form of encephalitis. Magnetic Resonance Imaging (MRI) plays a major role in the diagnosis of encephalitis and predicting the possible cause. This pictorial essay reviews the MRI findings of important types of viral encephalitis

Key Word: Herpes Simplex Encephalitis; Japanese Encephalitis, HIV Encephalitis, Dengue Encephalitis, Varicella zoster encephalitis, cytomegalovirus, MRI

[*]**Address for Correspondence:**
Dr. Rajeswaran R, Professor, Department of Radiology, Sri Ramachandra Institute of Higher Education and Research No.1, Ramachandra nagar, Porur, Chennai, Tamil Nadu 600116, INDIA.
Email: vanijayaraman58.om@gmail.com
Received Date: 04/04/2019 Revised Date: 22/05/2019 Accepted Date: 09/07/2019
DOI: https://doi.org/10.26611/10081118

Access this article online

Quick Response Code:

Website: www.medpulse.in

Accessed Date: 11 July 2019

INTRODUCTION

Viral encephalitis is the result of human virus affecting central nervous system. There are two types of encephalitis - Primary and secondary. Primary encephalitis is when the virus involves the brain and the spinal cord directly, while Secondary Encephalitis also called post infectious encephalitis occurs when infection spreads to brain from another part of the body MRI is the preferred modality for evaluation of encephalitis and it is useful for follow up. In this pictorial essay, we reviewed the MRI findings of patients with encephalitis referred to our hospital between Jan 2012 to Jan 2017 and subsequently proved on serology or CSF tests. All images were obtained from 1.5T MRI scanner. The protocol in our hospital includes obtaining: axial T1, T2, T2*, Diffusion Weighted images(WI) and Coronal or axial FLAIR images. Post contrast T1 WI obtained whenever needed.

DISCUSSION

Herpes Simplex Encephalitis (HSE): Herpes Simplex Encephalitis is the most common cause of fatal sporadic necrotizing viral encephalitis. The Incidence is one out of every 4 cases per million people. They are associated with high mortality and morbidity without proper treatment. There are two types described: Neonatal HSE and adult type. CSF and PCR values are usually gold standard for diagnosis, however MRI findings are considered important for aiding to diagnosis.

How to cite this article: Kalaivani J, Rajeswaran R, Prashanth J. Magnetic resonance imaging findings in viral encephalitis - A pictorial essay. *MedPulse – International Journal of Radiology.* July 2019; 11(1): 32-35. http://www.medpulse.in/Radio%20Diagnosis/

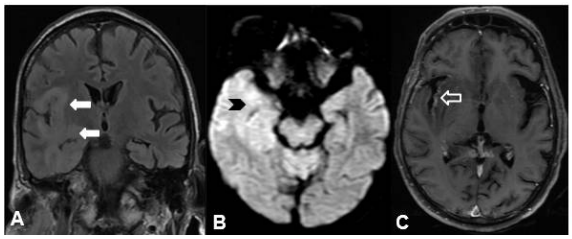

Figure 1

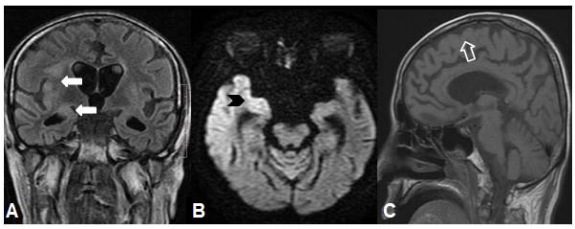

Figure 2

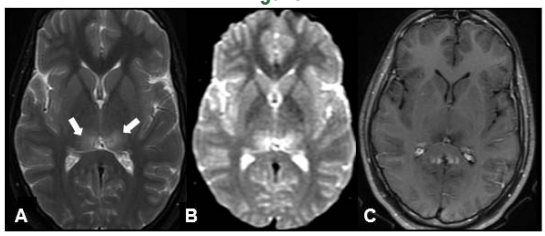

Figure 3

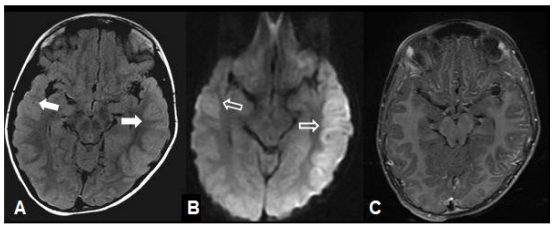

Figure 4

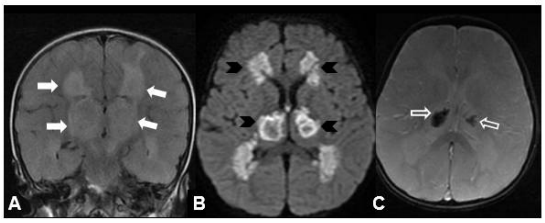

Figure 6

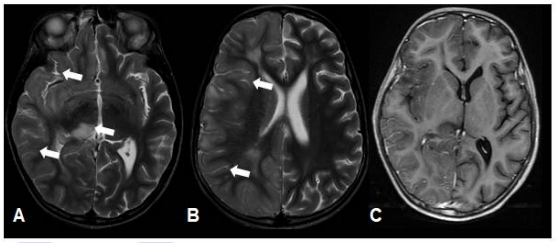

Figure 7

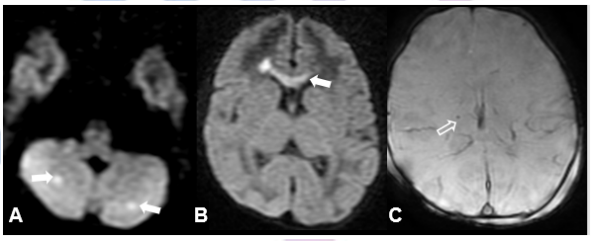

Figure 8

Legends

Figure 1: FLAIR coronal images (A) hyperintensity involving the right peri sylvian and temporal regions (Block arrows). DWI images (B) shows restricted diffusion in the right temporal region (black arrow head) . On contrast administration (C) mild enhancement is seen involving the sylvian fissure on the right side (Block arrows).

Figure 2: FLAIR coronal images (A and B) shows diffuse hyperintensities noted in bilateral periventricular white matter , thalamus (Block arrow) and caudate nucleolus (block arrow). DWI (C) images shows no evidence of restricted diffusion.

Figure 3: T2 coronal FLAIR (A) Images shows hyperintensity noted in the involving the cortex and sub cortical white matter of the right temporal region. DWI (B) image showing redistricted diffusion in the right temporal region. TI w sagittal (C) images of the patient showing diffuse cerebral atrophy.

Figure 4: T2W (A) image shows hyperintensity involving the bilateral posterior thalamus (Block arrow). On diffusion weighted images (B) there is mild restriction noted (Block arrow). Post contrast T1 images (C) no significant enhancement noted post contrast administration.

Figure 5: FLAIR images (A) shows hyperintensity in the bilateral temporal regions (Block arrow). DWI images (B) shows restricted diffusion in the bilateral temporal regions (Block arrow). Post contrast T1 images (C) shows no obvious enhancement following contrast administration.

Figure 6: Coronal FLAIR (A) images shows T2 hyperintensity with restricted diffusion (B) in the bilateral thalamus and peri ventricular white matter (Block arrow). On GRE (C) sequencing there is hemorrhage noted within bilateral thalami (Block arrow).

Figure 7: T 2 W (A and B) images shows hyperintensities in the right thalamus and the bilateral peri ventricular region (Block arrow). On T1 W post contrast (C) images shows no significant enchantment on post contrast images (Block arrow).

Figure 8: DWI (A and B) images showing restricted diffusion noted in the genu of the corpus callosum and bilateral cerebellar hemispheres (Block arrow)..

GRE sequence (C) shows blooming in the bilateral peri ventricular regions – may represent calcification (Block arrow).

Over 95% of the cases of HSE is caused by Herpes simplex virus (HSV)- 1. The infection is caused by gaining access through the nasopharyngeal mucosa. The viral gene remains latent within until any triggering event occurs like trauma, stress, immune suppression status or sometimes may occur spontaneously. Patients commonly present with fever, head ache, photophobia, generalized weakness, seizures.[1] MR imaging shows hypersensitivity involving the cortical and the sub-cortical regions on Fluid attenuated inversion recovery sequence (FLAIR)/T2 weighted images, bilateral symmetrical involvement of the temporal lobes and the insula being most common. There may be associated gyral swelling with loss of grey - white matter interface. Petechial hemorrhages are usually seen after 48 hours. Common differential diagnoses to be considered are infarcts and status epilepticus.[2] Status epileptics present unilaterally and typically involves mainly the cortex. Post ictal edema usually is more widespread and predominantly affects the entire hemispheric cortex. [3.]

Human Immuno Deficiency Virus (HIV) Encephalitis: HIV is a infection that has direct and indirect effect on CNS. Data show ~ 25% of the patients with HIV / AIDS infection will develop cognition impairments during the course of therapy. HIV initially affects the dendritic cells in the skin and Mucosa. It then binds to CD4 receptors in the langheran's cells; which then destroys and infects the CD4 positive T cells causing a burst of viremia which causes a wide spread tissue dissemination. The HIV infected cells can pass across the blood brain barrier and penetrate the brain. MRI shows volume loss with widening of the ventricle and sulci. Reduced grey white matter volume is seen in the medialfrontal gyri. White matter is near to normal on T1W Images and shows patchy bilateral symmetrical hyper intensity on T2WI / FLAIR Sequence. HIV lesions do not usually enhance on contrast or show restricted diffusion .[4] Differential diagnosis includes progressive multifocal leuko-encephalopathy [PML]. PML is an opportunistic infection caused by John Cunningham virus (JC virus). On imaging PML shows multifocal lesions commonly involving the posterior fossa white matter (more specifically the middle cerebellarpeduncle). Solitary lesions tend to present in the sub cortical U fibers. [5]

Japanese Encephalitis: The causative virus is spread by a vector and the disease is commonly found in South-Asian population; humans are accidental host in the disease. Patients present with symptoms like fever, rigors, head ache, focal neurological deficit and sometimes may be fatal. Patients who survive may be left with permanent neurological or psychotic sequelae. MRI characteristics are T2 / FLAIR hyper intensity in bilateral thalami. Unilateral involvement have also been reported, however to a far less extent. Other regions of involvement are basal ganglia, Substia nigra, red nucleus, pons, hippocampus, cerebral cortex and cerebellum. In some patients hemorrhagic transformation may occur, especially in thalami. There is usually no enhancement following contrast administration[6].Differential diagnosis to be considered is HSV encephalitis.

Dengue Encephalitis: Dengue is a single-stranded RNA virus belonging to the flavivirus genus. This causes dengue fever and Dengue hemorrhagic fever. Though Dengue is not clinically a neurotropic virus, Dengue encephalitis occurs due to neural infection by the virus. Analysis must be done whenever possible to isolate the virus or the antibody from the serum. Studies show that MRI after serology plays a major role in the diagnosis. MRI shows hyper intensity in globus pallidus on T2WI. Other sites that may be involved are hippocampus, temporal lobe and the pons. [7]

Varicella Zoster Encephalitis: The incidence of varicella - zoster has reduced since the introduction of varicella zoster vaccine in 1995. Despite the wide spread use of the vaccine, varicella - zoster continues to cause central nervous system disease. Varicella - zoster encephalitis has a median age of 40 -50 years at diagnosis and 30 % of the cases are under 15 years. It is commonly seen in immuno competent patients[8]. Symptoms are commonly seen 10 days after chicken pox rash or after varicella vaccination. Meningitis is seen in more than 50 % percent and encephalitis is seen in 40 % of the affected patients. MRI findings include cerebellitis with hyperintensities seen on T2W / FLAIR sequences. Restriction is usually seen on Diffusion weighted images. In varicella - zoster vasculopathy with stroke, T2 hyper intensities may be seen in cerebral cortex, basal ganglia and deep white matter. Contrast enhancement is patchy and mild in varicella - zoster encephalitis.

Creutzfeld Jacob Disease: Prion diseases, is a spectrum of neuro -degenarative disease that include Creutzfeld Jacob Disease [CJD], kuru, gerstmann - straussler - schenker syndrome and fatal familial insomnia. Out of these CJD is the most common to affect the humans (90 %), caused by proteinaceous infectious particles that are devoid of both DNA and RNA. They are four types of CJD namely: sporadic, familial, iatrogenic and variant. Clinical symptoms includes dementia, cerebral atrophy, myoclonic jerks, sensory and psychiatric symptoms, brownell oppenheimer variant (cerebellar syndrome) and heidenhain variant (cortical blindness). CJD affects the grey matter, more than the white matter. MR imaging shows T2/T2 flair hyperintensities in the basal ganglia, posterior thalamus (Pulvinar sign), posterior medial thalamus (Hockey stick sign).[9]

Rasmussen's Encephalitis: Though it is not part of the acute spectum discussed, it shows more of a chronic infection picture. It is a focal or localized encephalitis that shows clinical symptoms like epilepsy, hemiperises, altered sensorium. Patients affected are between 14 months - 14 years with peak incidence at 6 years of age. Exact etiology of Rasmuns Encephalitis is unknown. Viral infection or glutamate toxicity are the two possible etiologies. Though the initial MRI images do not show any finding, with passage of time hypersensitivity on T2W / FLAIR images are noted in the cortical and the sub cortical white matter of the affected hemisphere. There may be other areas affected like basal ganglia. Cortical atrophy is noted in late stages.[10]

Cytomegalovirus: Cytomegalovirus is one of the major cause of congenital infection in children. Out of these affected new borns only 10 % develop CNS manifestations. Clinically asymptomatic infants may show normal developmental milestones, whereas infants with clinical manifestations like Hepatosplenomegaly, petechiae, jaundice have worse prognosis. Hearing loss and delayed development are associated long term risk factors. Starting with a general rule that, the earlier the presentation the more-grave are the findings. MRI characteristics: The spectrum includes microcephaly with ventriculomegaly, white matter volume loss, delayed myelination and peri ventricular cysts. The abnormality in the cortex can extend from dysgenesis to lissencephalic pattern. T2 / FLAIR images show myelin delay or destruction and white matter volume loss. There may be associated confluent T2 hyperintensities with periventricular cysts. Periventricular calcifications with enlarged ventricles may also occur.

CONCLUSION

Each of the above discussed encephalitis has its own typical clinical presentation and MRI characteristics. A knowledge about them not only helps in diagnosis but also in the management.

REFERENCES

1. Egdell, Raymond, Darren Egdell, and Tom Solomon. "Herpes simplex virus encephalitis." BMJ: British Medical Journal (Online) 344 (2012).
2. McCabe, K., Tyler, K. and Tanabe, J., 2003. Diffusion-weighted MRI abnormalities as a clue to the diagnosis of herpes simplex encephalitis. Neurology, 61(7), pp.1015-1015.
3. Kramer, R.E., Lüders, H., Lesser, R.P., Weinstein, M.R., Dinner, D.S., Morris, H.H. and Wyllie, E., 1987. Transient focal abnormalities of neuroimaging studies during focal status epilepticus. Epilepsia, 28(5), pp.528-532.
4. Becker, J.T., Maruca, V., Kingsley, L.A., Sanders, J.M., Alger, J.R., Barker, P.B., Goodkin, K., Martin, E., Miller, E.N., Ragin, A. and Sacktor, N., 2012. Factors affecting brain structure in men with HIV disease in the post-HAART era. Neuroradiology, 54(2), pp.113-121.
5. Shah, R., Bag, A.K., Chapman, P.R. and Curé, J.K., 2010. Imaging manifestations of progressive multifocal leukoencephalopathy. Clinical radiology, 65(6), pp.431-439.
6. Siu, J.C.W., Chan, C.Y., Wong, Y.C. and Yuen, M.K., 2004. Magnetic resonance imaging findings of Japanese encephalitis. Journal-Hong Kong College Of Radiologists, 7(2), p.76.
7. Varatharaj, A., 2010. Encephalitis in the clinical spectrum of dengue infection. Neurology India, 58(4), p.585.
8. Pahud, B.A., Glaser, C.A., Dekker, C.L., Arvin, A.M. and Schmid, D.S., 2011. Varicella zoster disease of the central nervous system: epidemiological, clinical, and laboratory features 10 years after the introduction of the varicella vaccine. Journal of Infectious Diseases, 203(3), pp.316-323
9. Finkenstaedt, M., Szudra, A., Zerr, I., Poser, S., Hise, J.H., Stoebner, J.M. and Weber, T., 1996. MR imaging of Creutzfeldt-Jakob disease. Radiology, 199(3), pp.793-798.
10. Cece, H., Tokay, L., Yildiz, S., Karakas, O., Karakas, E. and Iscan, A., 2011. Epidemiological findings and clinical and magnetic resonance presentations in subacute sclerosing panencephalitis. Journal of International Medical Research, 39(2), pp.594-602.

Source of Support: None Declared
Conflict of Interest: None Declared