

# VIRAL ENCEPHALITIS: IMAGING FEATURES

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**Abstract :** Encephalitis commonly referred to as brain fever, implies inflammation of the brain. Since meningeal inflammation often accompanies this inflammatory process, term meningoencephalitis is sometimes used. Symptoms of encephalitis include sudden fever, headache, vomiting, weak muscles, impaired judgement and irritability. Encephalitis is usually caused by viral infection of the brain tissue but can also be caused by other factors such as exposure to certain bacteria, fungi, parasites, harmful chemicals or heavy metal poisoning. Herpes simplex encephalitis (HSE) is the commonest sporadic acute viral encephalitis all over the world. With few exceptions (for example, HSE), no specific therapy is available for most forms of viral encephalitis. The brain damage caused by acute encephalitis is due to a combination of intracellular viral growth and the host inflammatory response. Acute viral encephalitis is caused by direct viral infection of neural cells with associated perivascular inflammation and destruction of gray matter. Acute encephalitis constitutes a medical emergency. Early diagnosis is important for appropriate management. Magnetic resonance imaging of brain is the investigation of choice and the diagnosis may be confirmed by the polymerase chain reaction test for the virus in the cerebrospinal fluid. Mortality and morbidity may be high and long term sequelae are known among survivors. The emergence of some of the zoonotic encephalitis like Japanese encephalitis and Dengu encephalitis have posed an important public health problem in India. Encephalitis resulting from viral infection manifests as either acute viral encephalitis or postinfectious encephalomyelitis. Postinfectious encephalomyelitis follows infection with various viral or bacterial agents; the primary pathologic finding is demyelination of white matter, a non-infective inflammatory encephalitis that may require to be treated with steroids. In emergency cases, the presence of focal neurological signs and focal seizures will distinguish encephalitis from encephalopathy.

**Keywords:** Viral, Encephalitis, Imaging, MRI, CT

## VIRAL CAUSES

More than 100 different viruses can cause acute encephalitis. In India the most frequently reported causes are herpes simplex virus type 1 (HSV-1), arthropod-borne viruses (arboviruses) like Japanese encephalitis, Dengue, and Rabies caused by animal bites. In children herpes simplex virus type 2 (HSV-2), cytomegalovirus and measles are common sources of encephalitis. HIV is a common cause in all age groups.

**Table 1. Common viral causes of acute encephalitis in India<sup>1</sup>**

Cause	Characteristics of infection
Herpes simplex virus type 1	Common year-round; commonest cause of sporadic encephalitis in adults
Herpes simplex virus type 2	Common year-round; commonest cause of sporadic encephalitis in infants and children
Cytomegalovirus	Affects primarily immunosuppressed persons, particula children
HIV	Occurs year-round; affects all age groups
Measles	Occurs in winter and spring; commonly children
Japanese Encephalitis	Occurs in summer; all age groups
Dengue	Occur in winter; all age groups
Rabies	Occurs year-round; all age groups
Mumps	Occurs in winter and spring; parotitis, orchitis, common children
Rubella	Occurs in winter and spring; rash, lymphadenopath common in children
Varicella	Chickenpox or herpes zoster; all age groups

## OTHER DIAGNOSTIC CONSIDERATIONS

Differential diagnosis of encephalitis encompasses a large number of viruses as well as nonviral pathogens and noninfectious causes (table 2).

Patients with bacterial infection of the CNS usually appear more acutely ill than those with viral infection. However, meningitis caused by Streptococcus pneumoniae, Neisseria meningitidis, and Haemophilus influenzae type b may be insidious in onset. CNS infection caused by less virulent bacteria, such as Mycobacterium tuberculosis, Treponema

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**Table 2. Nonviral and noninfectious causes of encephalitis**

### Bacterial

Bartonella henselae  
Bartonella quintana  
Borrelia burgdorferi  
Brucella species  
Leptospira interrogans  
Listeria monocytogenes  
Mycobacterium tuberculosis  
Mycoplasma pneumoniae  
Rickettsia rickettsii  
Treponema pallidum  
Brain abscess or subdural empyema  
Partially treated bacterial meningitis

### Protozoal

Naegleria fowleri  
Acanthamoeba species  
Cysticercosis  
Echinococcus species  
Plasmodium falciparum  
Trypanosoma species

### Fungal

Blastomycosis  
Coccidioidomycosis  
Cryptococcosis  
Histoplasmosis

### Noninfectious

Central nervous system hemorrhage  
Collagen vascular disease  
Exposure to certain toxins or drugs  
Inborn errors of metabolism  
Malignant disease

pallidum, Borrelia burgdorferi, Bartonella henselae (cat-scratch disease) and Bartonella quintana also may be clinically indolent. Parameningeal bacterial infections (e.g., brain abscess, subdural empyema) may have features similar to those of viral CNS infections.

Other sources of encephalitis include noninfectious disorders such as metabolic derangement (e.g., hypoglycemia, electrolyte disturbances, uremia, hepatic encephalopathy, inborn errors

TABLE – 3 IMAGING FEATURES OF ENCEPHALITIS

S. No.	Type Of Infection	Location	T1 ↓=Hypointense ↑=Hyperintense	T2/FLAIR ↓=Hypointense ↑=Hyperintense	T1+C	Diffusion Restriction	T2* GRE	MRS ↓=decreased ↑=increased	Comments
1	HSV-1	Limbic System (Temporal & Inferior Frontal Lobes)	↓	↑	+	+	+	↓NAA/Cr ↑Cho/Cr	Involvement may initially appear unilateral but is typically followed by less severe contralateral disease. This "sequential bilaterally" is highly suggestive of herpes encephalitis
2	HSV-2	Diffuse & Nonfocal involvement. Initially periventricular white matter with relative sparing of the basal ganglia, thalami, and posterior fossa structures	↓	↑	+	+	-	nonspecific	No predilection to temporal & frontal lobes unlike HSV-1. Disease may progress to parenchymal calcification & cystic encephalomalacia
3	CMV	Periventricular White Matter	↓	↑	+	+	-	nonspecific	Subependymal enhancement is an infrequent finding but, if present, is a valuable diagnostic clue
4	Measles(SPE)	Posterior parietal, occipital and temporal regions, basal ganglia, corona radiata and subcortical and deep white matter	↓	↑	-	-	-	nonspecific	Nonspecific involvement in Acute & Subacute Measles Encephalitis  Basal ganglion involvement is noted in patients of SSPE who have severe disease and of longer duration
5	Varicella	Multifocal Areas of Cortex	↓	↑	-/+	+	-	nonspecific	A highly contagious disease with a generalized skin eruption; it usually occurs in childhood and is usually of little consequence in healthy children. However, may cause more serious disease, including encephalitis, in immunocompromised patients
6	Zoster	Brainstem/cortical GM, cranial nerves	↓	↑	-/+	+	-	nonspecific	Represents a reactivation of a latent varicella-zoster virus infection, usually in adulthood, can may have several CNS manifestations. Can be the causative agent in encephalitis, neuritis, and myelitis; it typically appears in patients with the characteristic skin eruption (shingles)
7	JE	Bilateral thalami, brainstem, cerebellum spinal cord, cerebral cortex	↓	↑	+	+	+	Absent NAA +Lactate +Choline +Creatinine	In a patient presenting with encephalitic illness from an endemic zone of JE with demonstration of lesions in the basal ganglia, thalami and brainstem should help in differentiating JE from atypical HSE.
8	Rabies	Brainstem, hippocampi, hypothalamus, WM, GM.	↓	↑	-/+	+	-	nonspecific	T1-Hyperintense bilateral BG is a rare but characteristic finding in appropriate clinical setting. Paralytic rabies can show Medulla and spinal cord T2- hyperintensity.
9	Dengue	Nonspecific involvement	↓	↑	-/+	+	+(In cases of JE coinfection)	nonspecific	There is paucity of radiological change in dengue encephalopathy/encephalitis, which is in contrast to JE where thalamic, basal ganglia and brainstem abnormalities are common. The MRI abnormalities includes oedema, scattered focal lesions and cerebral haemorrhage
10	HIV-1	Cerebral WM, brainstem, thalamus, BG	↓	↑	-	-	-	↓NAA ↑Cho ↑Myoinisotol	Cortical atrophy is the most frequent MR finding and is usually the only early alteration. The frontal lobes are the most common sites. White matter lesion in HIV encephalopathy are usually bilateral in asymmetrical manner. Gray matter is typically spared and mass effect is absent. The lesions do not enhance. A diffuse pattern of periventricular white matter hyperintensity suggests HIV encephalitis in contradistinction to the multifocal pattern produced by progressive multifocal leukoencephalopathy (PML)
11.	PML (caused by JC virus)	Periventricular and/or subcortical white matter	↓	↑	-	-	-	↓NAA ↓Creatinine ↑Cho ↑Myoinisotol(occasional) ↑Lipids/Lactate	Mimicks HIV encephalopathy. A multifocal distribution pattern is seen, which may be unilateral but is more often bilateral and asymmetric. There is an absence of mass effect and lesion rarely enhance.
12.	CJD (Creutzfeldt Jakob Disease) Slow virus disease caused by Prion infection	Corpus striatum, thalamus, cortical gray matter and occipital lobes	↓	↑	-	+	-	↓NAA	Symmetric abnormal, hyperintense T2 signal intensity within the corpus striatum is a relatively constant MR imaging feature. Diffusion-weighted images have been shown to be more sensitive in depicting involvement of the cortical gray matter or deep gray matter when compared with T2-weighted/FLAIR images. The hyperintensity seen on the diffusion-weighted images could be related to regions of restricted diffusion related to the intracellular vacuolation

of metabolism), malignant disease, collagen vascular disease, and intracranial hemorrhage. Exposure to certain drugs or toxins may also cause encephalitis.

## IMAGING FEATURES

Imaging is frequently nonspecific<sup>2</sup>. MRI with contrast is the investigation of choice. CT is frequently negative in early disease and underestimates the actual extent of the disease process.

Abnormal T2 hyperintensity of gray matter (GM) and deep grey nuclei is the best diagnostic clue in appropriate clinical setting. Concomitant white matter involvement are seen sometimes. Large, poorly delineated areas of involvement are common and patchy hemorrhages can be noted on T2\*GRE images. Most of the cases show restricted diffusion on Diffusion Weighted (DW) images reflecting presence of cytotoxic edema. Contrast enhancement is noted in most cases secondary to meningeal involvement. MR spectroscopy shows reduced NAA/Cr ratio and increased Cho/Cr ratio in cases of HSV-1 Encephalitis and presence of lactate in JE. No specific abnormality is noted on MR perfusion studies.

## DIFFERENTIAL DIAGNOSIS

Encephalitis is one of the important differentials of intraaxial brain masses<sup>3</sup>. These lesions include primary neoplasms (high- and low-grade), secondary (meta-static) neoplasms, lymphoma, tumefactive demyelinating lesions, abscesses, and **encephalitis**. Application of a diagnostic algorithm that integrates advanced MR imaging techniques (perfusion imaging, diffusion-weighted imaging, and MR spectroscopy) with conventional MR imaging findings may help the practicing radiologist make a more specific diagnosis for an intraaxial tumor. Intraaxial brain masses are a significant health problem and present several imaging challenges. The role of imaging is no longer limited to merely providing anatomic details. Sophisticated magnetic resonance (MR) imaging techniques allow insight into such processes as the freedom of water molecule movement, the microvascular integrity and hemodynamic characteristics, and the chemical makeup of certain compounds of masses.

## SPECIFIC VIRAL SYNDROMES

### *Acute Infectious Encephalitis/Herpes Simplex-1 Encephalitis*

HSV-I is a fulminant haemorrhagic meningoencephalitis involving the limbic system as a result of reactivation of latent viral infection, which spreads along the branches of the cranial nerves. HSV-1 is the cause of 95 percent of all herpetic encephalitis and the most common cause of fatal sporadic encephalitis. Clinical symptoms include an altered sensorium, a diminished level of consciousness, focal neurologic deficit and fever. CSF findings are non-specific.

HSV-I encephalitis has a particular predilection for the limbic system. Infection is therefore often localized to interomedial temporal lobes (fig. 1A), the orbital surface of the frontal lobes and insular cortex. There is sparing of the basal ganglia. Involvement may initially appear unilateral but is typically followed by less severe contralateral disease. This 'sequential

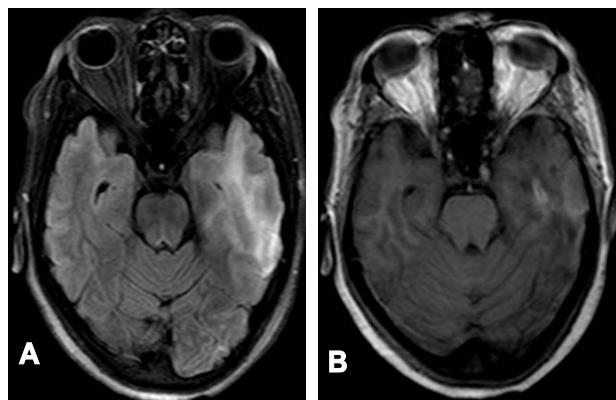


Figure 1: (A) FLAIR image shows hyperintensity in left temporal lobe. Subtle signal changes are also seen in right temporal lobe. (B) T1-weighted image showing hyperintensity in left temporal lobe suggestive of subacute hemorrhage.

'bilaterality' is highly suggestive of herpes encephalitis (see fig 1A). Hemorrhagic changes are common finding (fig 1B).

### *Herpes Simplex Type-2 Encephalitis*

HSV-2 is a major cause of neonatal encephalitis along with other TORCH agents (toxoplasmosis, other enteroviral infections, rubella, cytomegalic inclusion disease, herpesviruses, mumps, measles). The neonate typically becomes infected during birth, infection spreads from the mother to the child. Infection may lead to death or severe neurologic sequelae such as seizures, microcephaly, microphthalmia, ventriculomegaly, and multicystic encephalomalacia. Neuroimaging in this group of patients has not revealed the predilection for this virus to involve the temporal lobes, as seen in patients without AIDS, and imaging findings are typically diffuse.

### *Varicella-Zoster Virus*

The varicella-zoster virus can cause two seemingly different but actually closely related disorders, namely, varicella (chickenpox) and herpes zoster infection (shingles). Both disorders result in similar histopathologic findings. Varicella is a highly contagious disease with a generalized skin eruption; it usually occurs in childhood and is usually of little consequence in healthy children. However, varicella may cause more serious disease, including encephalitis, in immunocompromised patients. Herpes zoster represents a reactivation of a latent varicella-zoster virus infection, usually in adulthood. Varicella may have several CNS manifestations: cerebellar ataxia, meningoencephalitis, transverse myelitis, and aseptic meningitis. Herpes zoster can be the causative agent in encephalitis, neuritis, and myelitis. Neuroimaging findings are usually unremarkable. CT/MRI show multiple lesions in the deep white matter; haemorrhage may be seen.

### *Measles Encephalitis*

Measles is an acute, highly contagious infection caused by the rubeola virus, an enveloped RNA virus of the paramyxovirus family. There are three CNS syndromes related to measles infection acute measles encephalitis (AME),

subacute sclerosing panencephalitis (SSPE) and subacute measles encephalitis (SME).

AME is probably an autoimmune process and presents as fever during convalescence from measles. It is characterized by absence of the measles virus and by the presence of demyelination, necrosis, vascular damage and haemorrhage. No specific imaging feature has been described for it in literature.

SSPE is an invariably fatal neurodegenerative disease, developing as a sequel to early childhood measles infection. Following the original measles infection, the virus becomes altered and remains dormant intracellularly only to manifest as SSPE a decade or so later. Myoclonic attacks then develop and become increasingly more frequent, dementia follows and within months or a few years, the child is comatose and void of higher mental function.

SME usually occurs as an opportunistic infection in immunodeficient individuals with a latent period of 1 to 7 months. The prominence of cognitive and motor dysfunction is similar to that of SSPE but it has a subacute onset with more rapid evolution. The presence of seizures rather than myoclonus is the distinctive features of SME. MRI findings are non-specific.

#### **Acquired Immune Deficiency Syndrome (AIDS) and its related CNS Infections**

The retrovirus designated human immuno-deficiency virus (HIV) causes AIDS. Two HIV subtypes are recognized : HIV – I and HIV- 2. HIV-1 is the most widespread type and accounts for most AIDS cases. CNS involvement is both an early and a common feature of HIV infection.

HIV is a neurotropic virus that can directly involve both the peripheral and the central nervous system. The HIV virus itself is the most common CNS pathogen in AIDS patient followed by *Toxoplasma gondii* and *Cryptococcus neoformans*. The CNS infections in AIDS include.

- . HIV encephalitis – most common (60%)
- . *Toxoplasma* – commonest opportunistic infection (20-40%)
- . *Cryptococcus* (5%)
- . Progressive multifocal leukoencephalopathy PML (1-4%).

#### **Others**

- . Tuberculosis (2-18%)
- . Neurosyphilis (1 to 3%)
- . Varicella Zoster (< 1%)
- . CMV encephalitis

#### **HIV Encephalitis**

The most common neurologic complication seen in AIDS patients is subacute encephalitis. Clinical presentation includes a progressive dementia associated with motor and / or behavioural dysfunction.

The effects of cerebral HIV infection are clearly more evident on MR than CT. Cortical atrophy is the most frequent MR finding and is usually the only early alteration. T2- weighted images reveal hyperintense lesions in the periventricular white

matter and centrum semiovale that correspond to foci of demyelination and vacuolation. The frontal lobes are the most common sites. White matter lesion in HIV encephalopathy are usually bilateral in asymmetrical manner. Gray matter is typically spared and mass effect is absent. The lesions do not enhance. Fluid attenuated inversion recovery images are particularly useful in the detection of white matter disease. A diffuse pattern of periventricular white matter hyperintensity suggests HIV encephalitis in contradistinction to the multifocal pattern produced by progressive multifocal leukoencephalopathy (PML).

#### **Progressive Multifocal Leukoencephalopathy (PML)**

PML occurs in about 5 percent of patients with AIDS. Before the era of AIDS, PML was primarily associated with other immuno- deficient disorders, including renal transplantation, autoimmune disease, tuberculosis (TB), sarcoidosis, Whipple's disease, nontropical sprue and lymphoproliferative disorders. Patient treated with chemotherapy are also at increased risk for PML. Currently, PML appears to have a stronger association with AIDS than with any other immunosuppressive disorder and 55 to 85 percent of recent PML case are attributable to AIDS.

PML is a progressive demyelinating disorder arising from CNS infection with a papovavirus. The aetiologic agent is a human polyomavirus, the JC virus..

Clinical presentation of PML includes memory loss, visual deficit, personality changes, cognitive and speech disturbances, altered mental status, and motor and / or sensory abnormalities with a progressive neurologic decline.

#### **Cytomegalovirus (CMV) Encephalitis**

In adults, CMV is a frequent pathogen in the AIDS population and in other immunocompromised patients, occurring not only in the CNS but throughout the body. CMV more often presents outside the CNS, involving the respiratory tract, liver, gastrointestinal tract, genitourinary tract, and/or haematopoietic system. Neurologic manifestations of CMV include acute or chronic meningoencephalitis, cranial neuropathy, vasculitis, retinitis, myelitis, brachial plexus neuropathy and peripheral neuropathy.

In addition to atrophy. MR may demonstrate increased signal on T2-weighted images in the periventricular white matter, which may be patchy and is less often confluent. Infrequently, subependymal enhancement is evident and, if present, is a valuable diagnostic clue.

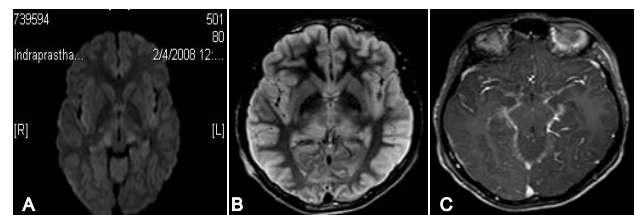


Figure 2: (A) Diffusion-weighted scan showing restricted diffusion in bilateral thalami. (B) Corresponding FLAIR images showing bithalamic hyperintensity. (C) T1-weighted contrast enhanced image showing mild meningeal (basal cistern) enhancement.

### **Japanese Encephalitis (JE)**

Epidemic of Japanese encephalitis (JE) occur in all countries throughout Asia. It usually appears in summer and is often severe with rapid progression to coma. The characteristic neurologic findings during the acute stage are extrapyramidal signs such as tremor, dystonia and rigidity. Seizures are more common in children than adults. It is important to distinguish JE from other types of encephalitis, particularly HSE, because antiviral therapy for HSE is very effective in the acute stage. Specific antiviral therapy is not available for JE, its treatment being supportive.

Diffuse meningoencephalitis affecting both gray and white matter of the cerebral hemispheres, basal ganglia, brainstem, cerebellum and thalamus has been reported<sup>4</sup>. Diffuse inflammatory changes are also found in the spinal cord, particularly in the anterior and lateral column.

### **Rabies**

Rabies encephalitis is an acute CNS infection caused by a type of RNA virus of the rhabdovirus family. The disease is also known as hydrophobia or aquifuga. Transmission to humans is mainly through bites of infected rabid dogs, cats, bats, and other wild animals. Human rabies may present in one of two forms: encephalitic and paralytic. In paralytic rabies, the medulla and the spinal cord are mainly involved by extensive neuronal damage and inflammation, whereas in the encephalitic form, it is the brain stem and the cerebrum, particularly the limbic system. Involvement of the basal ganglia and the thalamus is usually seen late in the disease.

Because the disease has a rapidly fulminant course, imaging of these patients is uncommon and difficult. Hence, the imaging findings in naturally acquired rabies have received little attention in the literature. A CT/MRI may show involvement of the basal ganglia, periventricular white matter, hippocampus, and brain stem<sup>5</sup>. Pontine hemorrhages have also been reported. Diffuse cerebral edema may be seen in advanced cases.

### **Dengue**

Dengue virus belongs to the family Flaviviridae, which also includes yellow fever, Japanese encephalitis (JE) and West Nile encephalitis viruses. The yellow fever infection presents with liver dysfunction and JE with encephalomyelitis. The dengue virus infection may manifest with both hepatic and neurological involvement. Neurological involvement in dengue can be attributed to metabolic alteration such as liver failure, electrolyte imbalance and renal impairment as well as hypotension. Bleeding and coagulation abnormalities in dengue haemorrhagic fever and dengue shock syndrome can also contribute to altered sensorium. The neurological complications of dengue virus infection include encephalitis, myelitis, Guillain Barre (GB) syndrome and myositis.

There is paucity of radiological change in dengue encephalopathy/encephalitis, which is in contrast to JE where thalamic, basal ganglia and brainstem abnormalities are common<sup>6</sup>. The MRI abnormalities include oedema, scattered focal lesions and cerebral haemorrhage.

### **Prion Infections : Creutzfeldt - Jakob Disease (CJD)**

Creutzfeldt-Jakob disease is a human spongiform encephalopathy. Like Kuru and Scrapie results form an

infection by a slow unconventional virus known as Prion. Dementia is usually the predominant feature of this disease, presenting in people between the ages of 40 and 80 years. The dementia may be accompanied by sensory abnormality, confusion and cerebellar ataxia. Prognosis is poor with a mean survival of less than 1 year from the onset of symptoms.

MR imaging clues in the diagnosis of Creutzfeldt – Jakob disease include progressive atrophic change of the brain or symmetric deep gray or cortical hyperintense lesion on the T2 – weighted images or diffusion images that lack gadolinium enhancement. Spectroscopy shows diminished levels of NAA.

### **Acute Disseminated Encephalomyelitis (ADEM)**

Acute disseminated encephalomyelitis is caused by immune response to a preceding viral infection or vaccination. Patients usually present with neurologic signs and symptoms 5 days to 2 weeks later. Both humoral and cell mediated immunity are implicated as the cause of pathologic changes. A hypersensitivity reaction to a myelin protein is thought to cause demyelination. Perivenous demyelination is the hallmark of the disease.

The disease primarily involves white matter, but change may also be apparent in gray matter and brainstem. CT may show low density in the periventricular white matter and MRI may show hyperintensity in the white matter on T2 weighted images and some of the lesions may exhibit contrast enhancement.

The *differential diagnosis* include multiple sclerosis, vasculitis and embolic infarction. In later stages of the disease, encephalomalacia, ventriculomegaly and atrophy may be seen.

### **SUMMARY**

Encephalitis affects persons in all age-groups and can result from a myriad of infectious and noninfectious causes. Depending on the cause, the outcome may be benign or in some cases fatal. History taking and physical examination can provide clues to the cause, but the diagnosis is usually established on the basis of CSF analysis, viral culture, MRI, and serologic testing, when indicated. MRI can help in the diagnosis of HSV-1 encephalitis, Japanese encephalitis, HIV encephalitis and Creutzfeldt Jakob's disease in appropriate clinical setting. However most of the time imaging is meant to rule out other possible causes in an acutely ill febrile patient showing focal neurological deficits and seizures. MRI is helpful in the assessment of complications and prognosis.

### **REFERENCES**

- (1) Kathleen M. Gutierrez, Charles G. Prober. Encephalitis. Identifying the specific cause is key to effective management. vol 103 / no 3 / March 1998 / Postgraduate Medicine
- (2) A Osborn, S Blaser, K Salzman, J Provenzale, Gregory L. Katzman, Mauricio Castillo, Gary L. Hedlund, Anna Illner, H. Ric Harnsberger, James A. Cooper, Blaise V. Jones, Bronwyn E. Hamilton : Diagnostic Imaging Brain. Amirsys Inc.
- (3) Riyadh N. Al-Okaili, Jaroslav Krejza, Sumei Wang, John H. Woo, Elias R. Melhem: Advanced MR Imaging Techniques in the Diagnosis of Intraaxial Brain Tumors in Adults. RadioGraphics 2006 : 26 : S173-S189
- (4) Kumar S, Misra UK, Kalita J, Salwani V, Gupta RK, Gujral R: MRI in Japanese encephalitis. Neuroradiology. 1997 Mar ; 39 (3):180-4.
- (5) Manasi Awasthi, Hemant Parmar, Tufail Patankar, Mauricio Castillo: Imaging Findings in Rabies Encephalitis. American Journal of Neuroradiology 22:677-680 (4 2001).
- (6) U.K. Misra, J. Kalita: Spectrum of Neurological Manifestations of Dengue in India. Dengue Bulletin – Volume 30, 2006.