Review Article

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Magnetic resonance imaging patterns of Viral Encephalitis: A pictorial review

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Abstract

Viral encephalitis is the infection of brain parenchyma caused by several groups of viruses. It can involve any age group and usually presents with the general symptoms of fever, headache, seizure, neurological deficit, or altered sensorium. However; few viruses can produce a specific group of systemic symptoms that can help to distinguish them from other causes. The two most important tools for determining the etiology of a disease are CSF examination and polymerase chain reaction (PCR). Furthermore, neuroimaging plays a critical role in diagnosing encephalitis, the direction and pattern of spread of the causative organism and the complications. In this pictorial essay, the various types of viral encephalitis are discussed along with their imaging pattern.

Keywords: Encephalitis, magnetic resonance imaging, viral encephalitis.

Introduction

Encephalitis primarily is of two types: Primary and secondary. Primary encephalitis includes infection of brain parenchyma and/or spinal cord directly as a result of viral infection. Secondary encephalitis includes the complications or the after effects occurring after the systemic manifestations like febrile seizure/status epilepticus-induced hypoxia, cerebral hypoperfusion secondary to shock, hepatic encephalopathy, electrolyte abnormalities, and leaky capillaries causing cerebral edema or PRES [1]. Neuroimaging plays a crucial role in directing towards the diagnoses alongside classic CSF examination and PCR. It can depict various specific patterns of spread as well as the non-specific findings like diffusion restriction, cerebral edema, hemorrhage, encephalomalacia, calcifications, necrosis etc. In this pictorial essay the aim is to:

- Access the role of MRI imaging in encephalitis.
- To recognize the pattern among various kinds of encephalitis which were further confirmed on CSF examination and PCR.

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DISCUSSION

1.Dengue fever encephalitis: In the new classification, the disease was classified as dengue without warning signs, dengue with warning signs, and severe dengue, with one definition for severe dengue as involvement of the central nervous

system (CNS) with impaired consciousness[2]. The various neurological manifestations of dengue fever may include encephalitis, encephalopathy, PRES like findings, ADEM like imaging features and hemorrhagic diathesis. The neurological complications of dengue fever may include encephalitis, encephalopathy, meningitis, ischemic and hemorrhagic stroke, cerebellar involvement, longitudinally extensive transverse myelitis (LETM), immune mediated syndromes, myositis, and neuro-ophthalmological disorders[3].

The encephalitis spread pattern of dengue fever involves basal ganglia, thalami, cerebellar structures, brainstem and cortical gray or subcortical white matter in isolation or combination. However, the classical site of involvement of the virus includes basal ganglia and/or thalami. The T2 hyperintensities in these areas usually show diffusion restriction. Dengue virus inflicts direct neuronal injury which usually involves bilateral basal ganglia and thalamus complex and manifests neuro-radiologically as "double-doughnut" sign (Figure 1). The "double-doughnut" sign is characterized by symmetrical T2/FLAIR high signal intensity area in bilateral thalami with restriction of diffusion on DWI and ADC and blooming in central region in gradient echo sequence due to hemorrhagic residues[4-7]. The patients may present with intracranial hemorrhages or microhemorrhages in case of hemorrhagic diathesis.

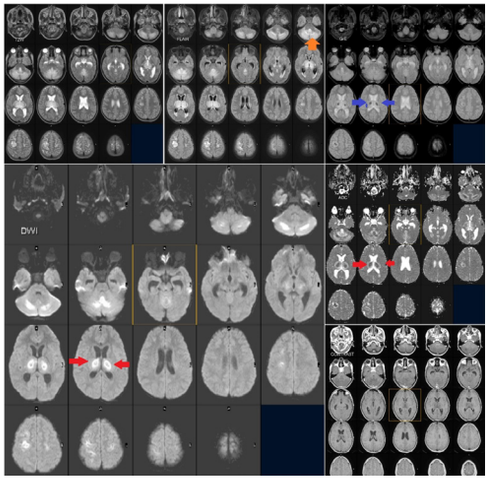

Figure 1: Dengue encephalitis- Axial T2, DWI, ADC, GRE, T1Wi & T2/FLAIR image showing hyperintense signal in bilateral thalami with central portion showing restricted diffusion (red arrow) and susceptibility of GRE images (blue arrow). T2/FLAIR hyperintense signal is also noted in cerebellar white matter (orange arrow)

2.Chikungunya encephalitis: Chikungunya is an arboviral disease which is transmitted by the Aedes mosquito. Neurological syndromes include encephalopathy, encephalitis, myelopathy, myelitis, Guillain-Barre syndrome, and neonatal hypotonia. Less frequently described features include sensorineural hearing loss, behavioral changes, and meningism [8].

The encephalitis pattern of spread includes involvement of discrete and confluent supra-tentorial T2W and FLAIR hyper-intense white matter foci, most of them showing diffusion restriction (Figure 2). Restricted diffusion in fronto-parietal white matter lesions is described as an early sign of viral encephalitis9.

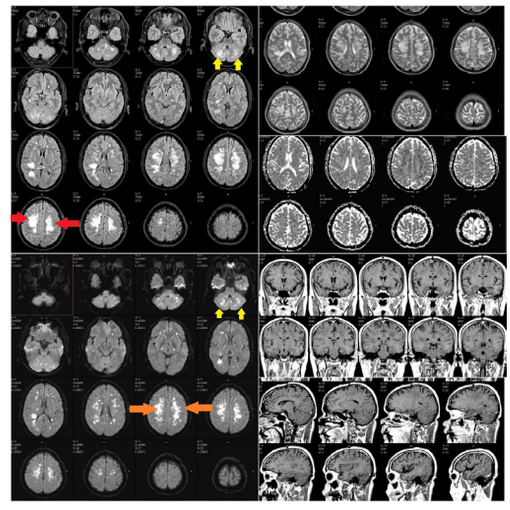

Figure2: Chikungunya encephalitis- Axial FLAIR, ADC, DWI, Post contrast coronal and sagittal T1Wi showing multiple discrete and confluent foci of T2 and FLAIR hyperintense signal involving cerebral white matter (red, orange arrow), cerebellar white matter (yellow arrow) and at bilateral middle cerebellar peduncle (blue arrow). Few of these lesions show tiny foci of restricted diffusion with no abnormal enhancement or post contrast T1Wi.

3.Herpes simplex virus (HSV) encephalitis: Patient usually present with seizure, altered sensorium and fever. HSV-2 is an important cause of neonatal encephalitis.

The encephalitis spread pattern includes restricted diffusion in supratentorial grey matter. It further includes three subtypes of spread patterns.

●First subpattern mainly involves neonates and children and involves scattered and asymmetric foci of restricted diffusion which suggest hematogenous spread of disease. Its differential includes septic emboli imaging specially in immunocompromised patients, however differentiating both entities is difficult [10]. Progression of lesions (within days) to form confluent areas of cortical/subcortical signal abnormality is suggestive of HSV encephalitis, whereas abscess formation is consistent with septic emboli [11,12].

●Second subpattern includes diffusion restriction areas in subcortical grey matter areas corresponding to the neural spread of the disease 10. In older children, cytotoxic oedema can affect the mesial temporal and insular cortices (Figure 3), relating to spread of viral particles along meningeal branches of the trigeminal ganglion [13].

●The third subpattern includes imaging findings of ischemic strokes in distinct vascular territories.

HSV infections are highly lethal and if not treated early can lead to cystic encephalomalacia and parenchymal calcifications [7].

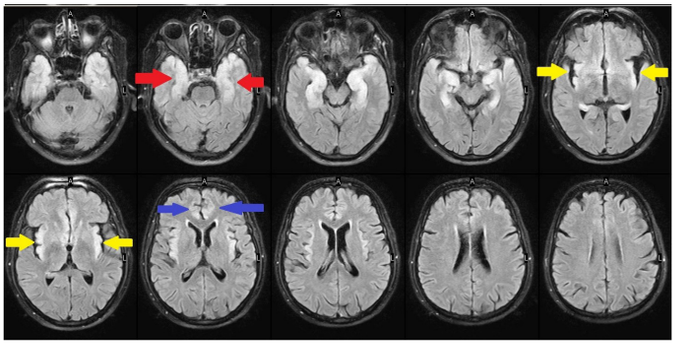

Figure 3: Herpes encephalitis-Axial T2 and FLAIR images showing symmetrical areas of hyperintense signal in bilateral medial temporal lobe, hippocampi (red arrow), insular cortex (yellow arrow) and cingulate gyrus (blue arrow).

4.Japanese encephalitis : Patients present with symptoms such as fever, rigors, headache, focal neurological deficit, and sometimes may be fatal. Patients who survive may be left with permanent neurological or psychotic sequelae [7].

Encephalitis pattern classically includes T2/FLAIR hyperintensities in bilateral thalami. Other structures to be involved are basal ganglia, substantia nigra, red nucleus, pons, hippocampus, cerebral cortex, and cerebellum (Figure 4). In some patients, hemorrhagic transformation may occur, especially in thalami. There is usually no enhancement following contrast administration [14].

Treatment includes giving supportive empirical therapy.

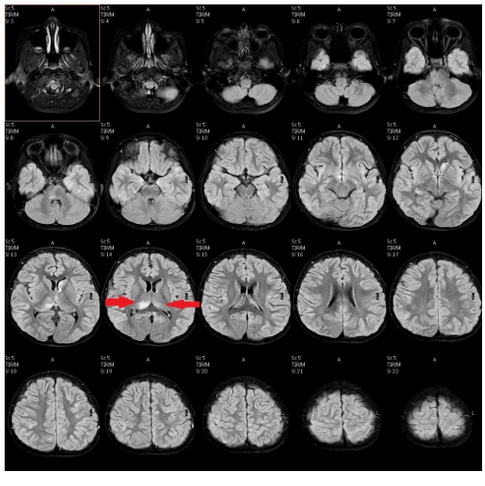

Figure 4: Japanese encephalitis- Axial FLAIR images showing asymmetrical hyperintense signal in bilateral thalami (red arrow).

5.Covid-19 encephalitis: The patient presents with respiratory distress, fever, headache, mental confusion and altered sensorium. The imaging features involve confluent FLAIR hyperintensities involving the gray & white matter, microhemorrhages, and leptomeningitis (Figure 5,6). The lesions majority of the times shows diffusion restriction. The other common areas to get involved are basal ganglia, corpus callosum specially splenium and pons.

Symmetric confluent leukoencephalopathy favors a component of post–hypoxic leukoencephalopathy, and more focal WM lesions (particularly if the spinal cord or the posterior fossa is involved) may suggest post-viral autoimmune demyelination [15].

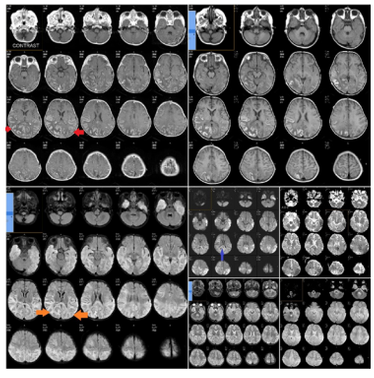

Figure 5: COVID encephalitis-Axial DWI, ADC, FLAIR , T2W and pre/post contrast T1W images are showing patchy areas of restricted diffusion in bilateral cerebral white matter and splenium of corpus callosum (blue arrow) with FLAIR hyperintense signal (orange arrow). Areas of hemorrhages seen as T1 hyperintense signal noted within the bilateral parieto-occipital cortex and subcortical whit matter (red arrow).

6.Enterovirus encephalitis: The patients may initially present with gastrointestinal symptoms or rash followed by neurological involvement and features of rhombencephalitis.

The typical pattern of spread includes involvement of brainstem or the cerebellum. When enterovirus causes rhombencephalitis, MRI typically demonstrates T2 hyperintensity of the dorsal pons and medulla oblongata, and there may be involvement of the midbrain, dentate nuclei and upper cervical cord (Figure 7) (serotypes E-71 and E-68 are often cited as the causative agent) [16-17].

The other causative agents causing similar imaging pattern may include Burkholderia, rotavirus and Listeria monocytogenes.

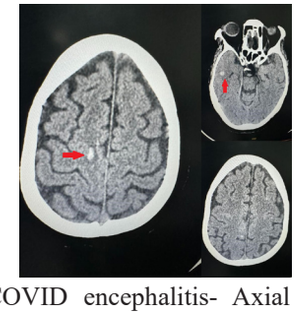

Figure 6: COVID encephalitis- Axial CT images showing multiple tiny foci of petechial hemorrhage at gray white matter junction in cerebral parenchyma(red arrow) .

7.Rotavirus encephalitis: The patients present with gastroenteritis, convulsions, encephalopathy or encephalitis. The rotavirus cerebellitis is the most classical manifestation whereas cerebellar mutism is the most interesting finding although can be reversible. The imaging features vary according to the course of the disease.

The MRI findings include transient T2/FLAIR hyperintense lesions with diffusion restriction in white matter which may progress further or may resolve followed by involvement of cerebellum (Figure 8A). Cerebellar edema (usually bilateral) is seen followed by cerebellar atrophy in later stages[10].

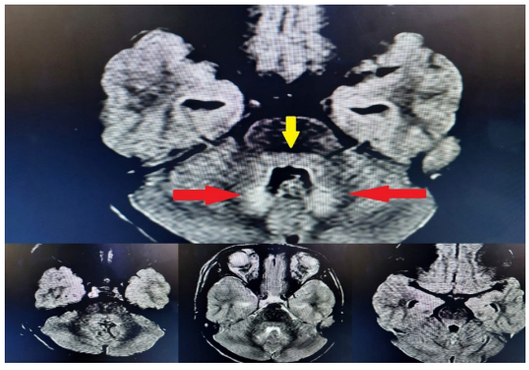

Figure 7: Enterovirus encephalitis- Axial DWI, FLAIR and T2Wi showing hyperintense signal involving dorsal portion of pons (yellow arrow), extending into bilateral medial cerebellar white matter (red arrow).

8.Rabies encephalitis: The disease is fatal most of the times with the patients presenting with variety of symptoms like altered sensorium, convulsions, hyperreflexia or fever with common history of dog bite. The confirmatory diagnosis is the isolation of virus from biological samples or detection of the rabies antigen or antibodies, MRI can be helpful in identifying early features. The predilection of rabies is for brainstem, thalami and hippocampi.

MRI findings in encephalitic form and paralytic form show ill-defined hyperintense lesions in the brainstem involving the dorsal aspect of the medulla, pontine tegmentum, periaqueductal gray matter, collicular plate, as well as the central white matter of the midbrain, deep and cortical gray matter, deep and subcortical white matter, hippocampi, medial aspects of the thalami, and in the hypothalamus on both sides of midline on T2W imaging (Image 8B) 18-19. There is also role of DW/ADC maps which show increased diffusion in rabies encephalitis. MRI findings in paralytic rabies can show hyperintense signal in medulla extending to cervical cord associated with cord expansion [20].

Contrast-enhanced studies do not reveal enhancement of these structures in the early phase, while mild-to-moderate enhancement of the hypothalamus, brainstem, and gray matter of the cord may be seen when the patient becomes comatose. The brachial plexus is an exception and can show enhancement in the early prodromal phase of the disease 18. There are no microhemorrhages seen.

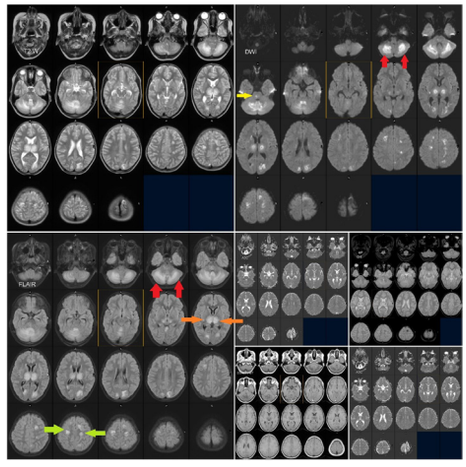

Figure8 A: Rotavirus encephalitis- Axial T2, DWI, ADC, GRE, FLAIR and T1Wi showing hyperintense signal involving bilateral thalami (orange arrow), cerebellar white matter (red arrow), dorsal portion of pons (yellow arrow) and cerebral white matter (green arrow).

Note is made of absence of susceptibility within the signal in bilateral thalami on GRE images which was noted in dengue encephalitis.

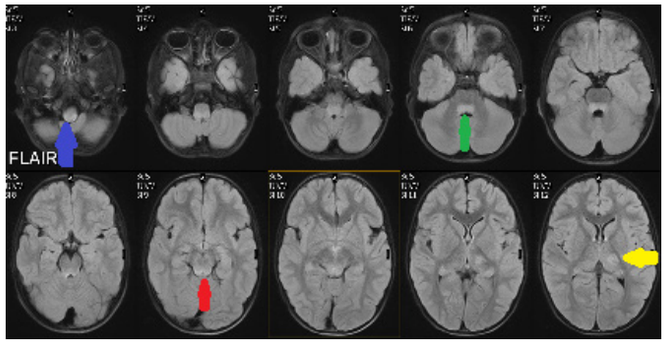

Figure8 B: Rabies encephalitis- Axial FLAIR images showing hyperintense signal in posterior portion of midbrain (red arrow), pons (green arrow), medulla (blue arrow) & within left thalamus (yellow arrow)

9.HIV Encephalitis: HIV enters the CNS within early phase of infection and cause chronic CNS manifestations. Even after starting ART treatment, HIV continues to replicate since many drugs do not cross the blood brain barrier. Patient manifest neurocognitive symptoms in late stages of the infection.

MRI in early stages does not show any significant findings, therefore if any significant finding is seen, other disorders must also be considered. The signal changes in later HIV encephalopathy stages involve T2 /FLAIR hyperintense

and T1 iso/hypointense signal in bilateral deep white matter and rarely can be seen in subcortical structures and brainstem. This stage often progress with dominant cerebral brain atrophy. No enhancement of affected areas are seen on post contrast images (Figure 9).

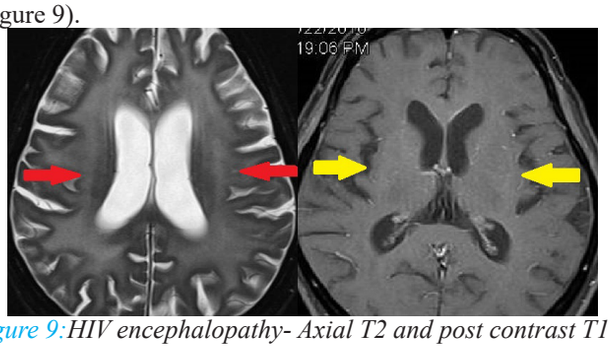

Figure 9:HIV encephalopathy- Axial T2 and post contrast T1Wi showing diffuse T2 hyperintense signal in cerebral white matter without abnormal enhancement.

10.HIVrelated- Progressive Multifocal Leukoencephalopathy: It is progressive demyelinating disease caused by JC virus. It is usually reactivated in immune-compromised patients. It presents as focal neurological deficits and the patient's condition worsens by day due to progressive demyelination.

The MRI findings of PML show asymmetric more commonly than symmetric demyelinating lesions appearing hyperintense on T2 / FLAIR and hypointense on T1Wi. Contrast enhancement is usually absent however the lesions may show peripheral enhancement sometimes. The lesions usually initially appear in subcortical white matter, including U fibres. The newer, spreading demyelinating margin is called the frontline, whereas older portions of the lesions show advanced demyelination. The frontline margins on T2-weighted images show a diffuse pale hyperintensity and/or numerous discrete hyperintense dots giving a milky way appearance[21]. This often corresponds to the hyperintense rim on DWI. In many cases, the hyperintense rim on DWI is interrupted at older demyelinated margins. With progression, the intensity gradually increases on T2-weighted images and coalesces to form a homogeneous advanced demyelinated lesion of near-CSF intensity[22]. MR spectroscopy show mildly elevated choline & reduced NAA and therefore should not be confused with low grade glioma (Figure 10).

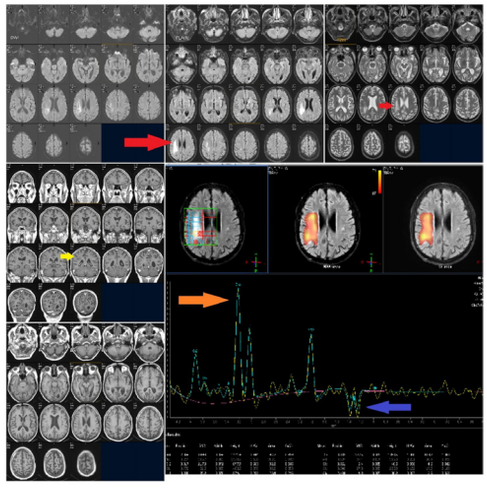

Figure 10:PML HIV—Axial DWI, ADC, FLAIR, Post contrast axial and coronal T1 and MRS images showing well defined large T2 and FLAIR hyperintensefocus (red arrow) involving deep cerebral white matter in right corona radiata without abnormal enhancement on post contrast T1Wi (yellow arrow). MRS image shows mildly elevated choline peak (orange arrow) and lactate peak (blue arrow).

11.Epstein-Barr virus (EBV) encephalitis: EBV can cause encephalitis, a rare but serious complication of primary or reactivated EBV infection. Clinically, EBV encephalitis presents with a range of symptoms, including fever, altered mental status, seizures, and focal neurological deficits. Patients may exhibit confusion, irritability, and motor abnormalities. In severe cases, coma or respiratory failure can occur[23]. Radiological imaging, particularly MRI, plays a crucial role in diagnosing EBV-associated encephalitis. Commonly affected areas include the cortex/subcortex (image 11) , white matter and basal ganglia . Other areas like the thalamus, brainstem, substantia nigra, and cerebellum may also show involvement. Although brain abscesses and hemorrhages are rare, these can be seen in some patients, particularly in those with immunocompromised states. The imaging pattern in EBV encephalitis typically features T2 hyperintense lesions and may show enhancement after contrast administration. Prognosis correlates with the affected regions; isolated hemispheric involvement often results in a better outcome, while brainstem or thalamic involvement is associated with poorer recovery[24].Diagnosis is supported by detecting EBV DNA in the cerebrospinal fluid (CSF) via PCR. Early recognition and supportive care are critical for improving patient outcomes.

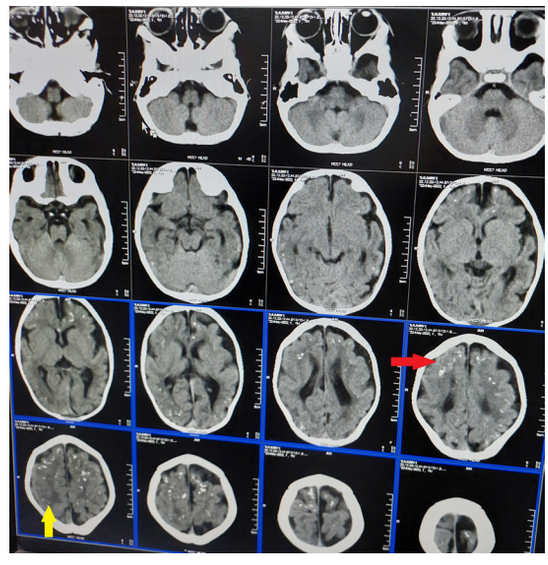

Figure11: EBV- Axial CT images in a case of perinatal EBV encephalitis shows multiple calcifications at grey white matter junction (red arrow) with white matter hypodensity (yellow arrow). There was associated microcephaly.

12.Swine flu encephalitis: The symptoms occur mostly in few days of infection and show almost complete recovery within a month. Major MRI findings include T2/FLAIR hyperintense signals involving thalami, cerebellar hemispheres, brain stem, and centrum semiovale bilaterally. Symmetric splenial lesions (restricted diffusion and T2 hyperintensities) of the corpus callosum have been frequently reported. These lesions show restriction diffusion. There is specific predilection for involvement of perirolandic region (Figure 12)[25].

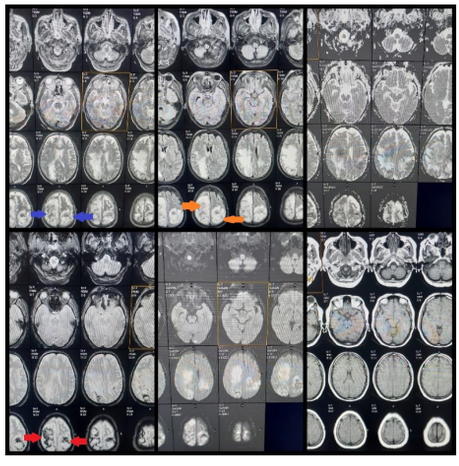

Figure 12: Swine flu encephalitis- Axial T2, FLAIR, ADC, T1, DWI and GRE images in clockwise direction showing multiple T2 and FLAIR hyperintense foci involving bilateral fronto-parietal white matter and overlying cortex with patchy areas of restricted diffusion (blue, orange arrow). Areas of restricted diffusion (blue, orange arrow). Areas of hemorrhage noted in the affected area in perirolandic location on GRE images as foci of susceptibility (red arrow).

The other common patterns seen in neurological manifestation of viral infection include: A.Acute Disseminated Encephalomyelitis(ADEM):ADEM is immunologically mediated inflammatory response typically occurring post infection or post vaccination. It generally manifests in pediatric population. Patients usually present with fever, malaise, headache, nausea, or vomiting and then occasionally symptoms progressing to coma or decerebrate posturing. For encephalopathy to be diagnosed, altered mental status or behavioral change must be present.

The typical MRI findings described in ADEM are widespread, bilateral, asymmetric patchy areas of homogeneous or slightly inhomogeneous increased signal intensity on T2-weighted imaging within the white matter, deep gray nuclei, and spinal cord (Figure 13,14).

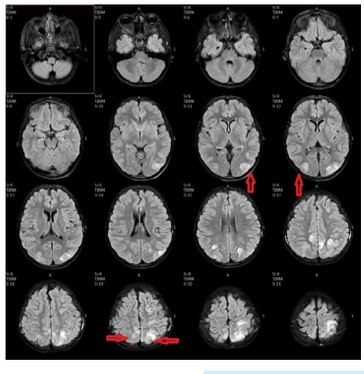

Figure13: ADEM-Axial FLAIR images showing multiple large areas of hyperintense signal involving subcortical white matter and overlying cortex (red arrow).

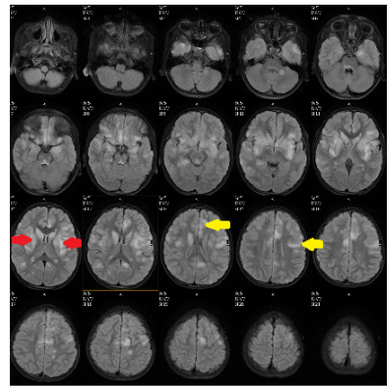

Figure14: ADEM- Axial FLAIR images showing multiple large areas of hyperintense signal involving subcortical white matter ,overlying cortex (yellow arrow) with involvement of bilateral deep nuclei (red arrow) .

Within the white matter, juxtacortical and deep white matter is involved more frequently than is periventricular white matter, which is an important contrast to patients with multiple sclerosis. Infratentorial lesions are common, including the brainstem and cerebellar white matter. These lesions typically appear simultaneously with clinical presentation [26].

B.Acute encephalopathy with biphasic seizure and late restricted/reduced diffusion:It usually has influenza as common etiology and show biphasic clinical presentation. In early phase convulsive febrile seizure occur which are followed by reduced conscious state, with or without persisting fever. In this early phase MRI findings are usually normal. The second phase occurs at day 4-6 into illness characterized by seizures recurrence with or without consciousness. The MRI imaging findings during this phase after the secondary seizures include T2/FLAIR hyperintensities involving subcortical U fibres showing true restriction diffusion, sparing perirolandic regions (Figure 15). Recovery occurs within weeks to months.[27, 28]

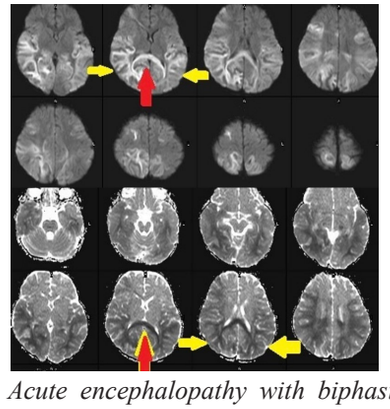

Figure 15: Acute encephalopathy with biphasic seizure and late restricted/reduced diffusion- Axial DWI and ADC images showing areas of restricted diffusion involving splenium of corpus callosum (red arrow) and bilateral cerebral white matter in temporo-parieto-occipital lobe (yellow arrow).

C.Acute Necrotizing Encephalopathy of Childhood (ANEC):It is mostly the complication of post-viral infections. Most commonly, it manifests in pediatric population, however can be seen sometimes in adults also.The MRI imaging findings include T2/FLAIR hyperintense lesions involving bilateral thalami, brainstem, cerebral white matter, and the cerebellum. Diffusion restriction is present (Figure 16).Hemorrhagic transformation may occur with contrast rim enhancement[29].

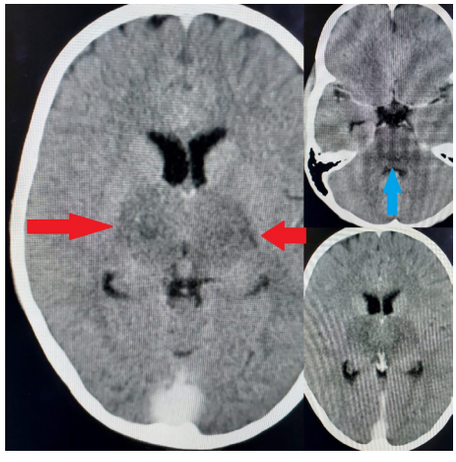

Figure 16: Acute necrotizing encephalitis- contrast axial CT images showing non enhancing hypodense areas involving bilateral thalamus (red arrow) and pons (blue arrow) with central area of necrosis / liquefaction.

13.Creutzfeldt-Jakob Disease Encephalitis : Creutzfeldt-Jakob disease (CJD) is not seen in pediatric age group however to complete the list of various viral encephalitis , we have added about this as an addendum. It is the most common prion disease and is universally fatal. It typically presents with rapidly progressive dementia. Early MRI findings show spongiform changes, with vacuole formation in the gray matter that causes diffusion restriction, leading to hyperintense signals on diffusion-weighted imaging (DWI), more prominent than on T2-FLAIR. As the disease progresses, gliosis becomes more pronounced, and hyperintensity on T2-FLAIR becomes more significant than on DWI (Figure17).

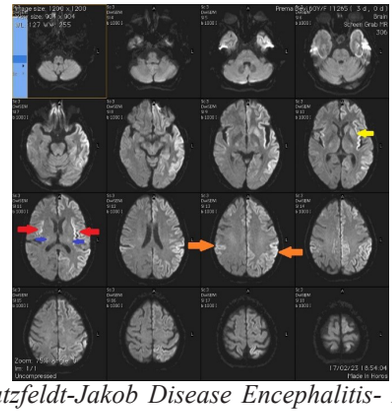

Figure 17:Creutzfeldt-Jakob Disease Encephalitis- DWI axial images showing hyperintense signal involving bilateral basal ganglia (yellow arrow), thalami (blue arrow), insular cortex (red arrow) and cerebral cortex (orange arrow).

Affected areas are often bilaterally symmetrical but can be asymmetrical. Commonly involved regions include the insula, cingulate gyrus, superior frontal gyrus, and the striatum30. Several variants of CJD present with distinct patterns of brain involvement: the Brownell-Oppenheimer variant affects the cerebellum and basal ganglia, often seen later with atrophy; the Heidenhain variant initially involves the parieto-occipital cortex; and the Stern-Garcin variant affects the basal ganglia and thalamus. Thalamic involvement in CJD, particularly in the variant form, is characterized by the "Hockey Stick" or "Pulvinar" sign—bilateral hyperintensity of the pulvinar and dorsomedial thalamic nuclei on FLAIR imaging, resembling a hockey stick shape. This distinctive MRI feature helps differentiate variant CJD from other prion diseases[31].

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