Original Article

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Radiology Section

Role of MR Imaging in Viral Encephalitis-A Clue Towards Aaetiological Agent

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ABSTRACT

Introduction: MRI plays a significant role in diagnosing viral encephalitis. It aids the diagnosis of causative agent, conditions mimicking the disease, identification of complications and hence prognostication of the patient. The present study analysed the MRI spectrum of different types of viral encephalitis and its role in diagnosing the causative virus.

Aim: To study MRI findings of viral encephalitis and their correlation with CSF/biochemical/histopathological microbiological analysis.

Materials and Methods: All patients suspected or previously diagnosed of CNS infections and referred to the department of Radiodiagnosis at Dayanand medical college and hospital, Ludhiana for imaging over a period of one and a half years were included in this study.

Results: Most of the patients presented with fever and altered sensorium. CSF showed mildly raised proteins and pleocytosis. Asymmetric involvement of medial temporal lobes, cingulated gyri and insular cortices in herpes simplex infection was classical. Thalamic haemorrhages with/without basal ganglia and brainstem involvement were seen in dengue encephalitis and Japanese encephalitis. Diffusion restriction and haemorrhage were not seen in rhombencephalitis due to rabies. Varicella zoster vasculopathy involved both large and small vessels. Post viral demyelination [Acute Demyelinating Encephalomyelitis (ADEM)] mimicked viral encephalitis on MRI. An antecedent history of fever and response to steroids favoured the diagnosis of ADEM.

Conclusion: We concluded that MRI is the modality of choice for diagnosing viral encephalitis and suggesting specific aetiological virus in an appropriate clinical and biochemical setting.

Keywords: Demyelination, Encephalitis, Hyperintense, Magnetic resonance imaging, Vasculopathy

INTRODUCTION

Central nervous system (CNS) involvement by viral infections in humans is not uncommon and is seen predominantly in children or immune-compromised individuals. Viruses can involve brain parenchyma, meninges and spinal cord in isolation or in combination and may cause life threatening complications.

Unlike bacterial and fungal infections of brain, MRI findings of viral encephalitis are quite suggestive of the type of virus in the presence of appropriate clinical setting and CSF pleocytosis. Vasculopathy, involvement of specific areas, haemorrhagic lesions and diffusion restriction are some of the pointers towards diagnosis. As with all CNS infections, magnetic resonance imaging (MRI), with its superior contrast resolution is the investigation of choice for imaging viral encephalitis over computed tomography (CT) [1].

Objectives

1. Analysis of magnetic resonance imaging findings in viral infections of CNS.
2. To correlate the neuroradiological diagnosis with CSF/biochemical/histopathological/microbiological analysis.

MATERIALS AND METHODS

This longitudinal prospective study was conducted over a period of one and a half years from August 2015 to January 2017 with a study population of 75 patients in the Radiodiagnosis department of Dayanand Medical College and Hospital, Ludhiana, India.

All patients diagnosed of or clinically suspected of CNS infections were subjected to MRI in the department. MRI was performed on MAGNETOM Avanto 18 Channel 1.5 Tesla TM MR Machine (Siemens India Ltd.). The institutional ethical committee accorded ethical clearance to this study.

Informed consents and detailed clinical history were taken from all the patients/guardians before the examination. Neurological examination findings were also recorded.

Inclusion Criteria

a) All cases suggestive of viral or postviral CNS infections.

b) The diagnosis of viral encephalitis was suspected based on the history of fever and presentation with altered sensorium with or without focal neurological deficit or seizure.

c) Patients with CSF pleocytosis and mildly raised CSF protein were taken.

Exclusion criteria

a) Tubercular, bacterial or fungal meningitis were excluded.

b) Patients with generalised contraindications for MR imaging such as patients with cardiac pacemakers, neurostimulators, non-compatible prosthetic implants, claustrophobia etc.

c) Patients with brain tumours and infections other than viral aaetiology.

Preparation of Patient

The patients were instructed to remove all metallic objects such as jewelry, keys, credit cards, watches, coins etc., before examination. Expected time of scan and the procedure was also explained to all the patients. During the scan, patients were in contact with the technician/doctor by a two-way intercom system.

MR Protocol

The patients were positioned in the MR scanner and localizers were taken in all three planes (axial, coronal and saggital).

Turbo spin echo T2W sequence [(TR) (TE)/number of excitations (n)=4050 ms/101 ms/3], Spin echo (SE) T1W sequence (TR/TE/n=652 ms17 ms/1), FLAIR-Fluid attenuated inversion recovery sequence (TR/TE/n=9000 ms/1; inversion time-2500ms), Contrast enhanced T1W sequence, GRE sequence (TR/TE= 761 ms/26 ms), diffusion weighted (DW) imaging using echo planar imaging (EPI) sequence with TR/TE =3500 ms/109 ms (minimum) and diffusion sensitizing gradients were applied with b=0, 500 and 1000s/mm^2.

STATISTICAL ANALYSIS

MRI findings on various sequences like T1W, T2W, FLAIR, GRE, Diffusion weighted and Contrast enhanced scan were recorded as per proforma. The data was analysed using descriptive statistics.

RESULTS

We analysed the neuroimaging features of 75 cases that included viral encephalitis and post viral demyelination ADEM. It was observed that children in the first decade were involved the most (36.36%) followed by middle aged adults between 21-50 years. CSF analysis was done in 69 cases, which showed raised mean CSF protein levels (61.1 mg/dL), normal mean glucose levels (71 mg/dL) and pleocytosis. Number of cells per tap varied from 2 to 450 with an average value of 47, which were predominantly lymphocytes. Most of the patients presented with fever and altered sensorium (49 patients), which in most patients occurred concurrently with other symptoms like headache, seizures, abnormal body movements, focal weakness etc. Imaging features varied in different viral infections. The anatomical localisation of focus of disease and certain MR features helped in characterisation for the specific viral agent [Table/Fig-1,2].

Imaging Parameter	No. of Patients	Percentage
Leptomeningeal/Pachymeningeal involvement	10	13.33%
Temporal lobe involvement	25	33.33%
Frontal and other lobar involvement	29	38.66%
Basal ganglia	18	24.00%
Thalamus	22	29.33%
Brain stem involvement	16	21.33%
Cerebellar involvement	12	16.00%
Presence of T2/FLAIR hyperintensity	58	77.33%
Presence of diffusion restriction	24	32.00%
Presence of haemorrhage	13	17.33%
Presence of Hydrocephalous	None	
Presence of infarcts	22	2.94%
Associated cord involvement	6	8.00%

[Table/Fig-1]: Imaging analysis of cases with viral infection of the central nervous system (n=75).

Aetiology	Number of cases	Percentage
Herpes	18	24.00%
Japanese encephalitis	11	14.66%
Dengue encephalitis	4	5.33%
Rabies encephalitis	2	2.66%
ADEM	15	2.00%
CMV	5	6.66%
Herpes zoster	9	12.00%
Non specific viral encephalitis	7	9.33%
False positive (Reye's encephalopathy)	4	5.33%

[Table/Fig-2]: Distribution of causes of viral infection of the CNS (n=75).

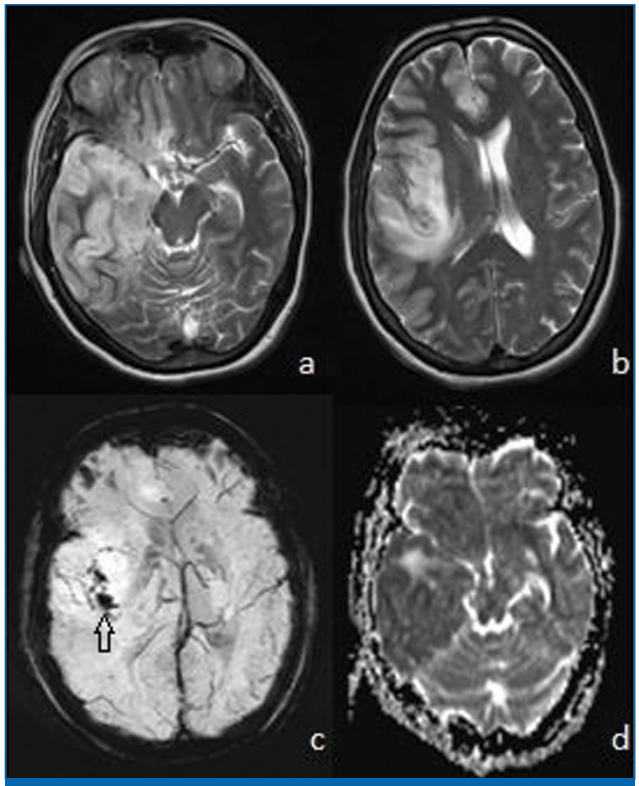
DISCUSSION

We analysed the neuroimaging features of 75 cases that included viral encephalitis and post viral demyelination (ADEM).

18 patients were diagnosed of Herpes encephalitis based on their clinical features, neuroimaging, CSF analysis and response to anti-herpes antiviral drugs. CSF was normal in six of them, which is a well established fact [2].

Insular cortex involvement in Herpes encephalitis was the most common and prominent feature, seen in 17 out of 18 patients. Asymmetrical and bilateral T2 hyperintense signal was seen involving the cortex and underlying white matter in medial temporal lobes (in 13 patients) and cingulate gyri (in 15 patients). None of them had lesions limited to the hippocampus. Distribution of lesions of Herpes encephalitis in the temporal lobes, cingulate gyri, orbital surfaces of frontal lobes and insular cortices as seen in our cases [Table/Fig-3] is quite specific and has been described in the past too [3].

Schroth G et al., opined that lesions strictly limited to hippocampus should prompt the consideration of diagnosis other than Herpes encephalitis, as observed in our study [4].

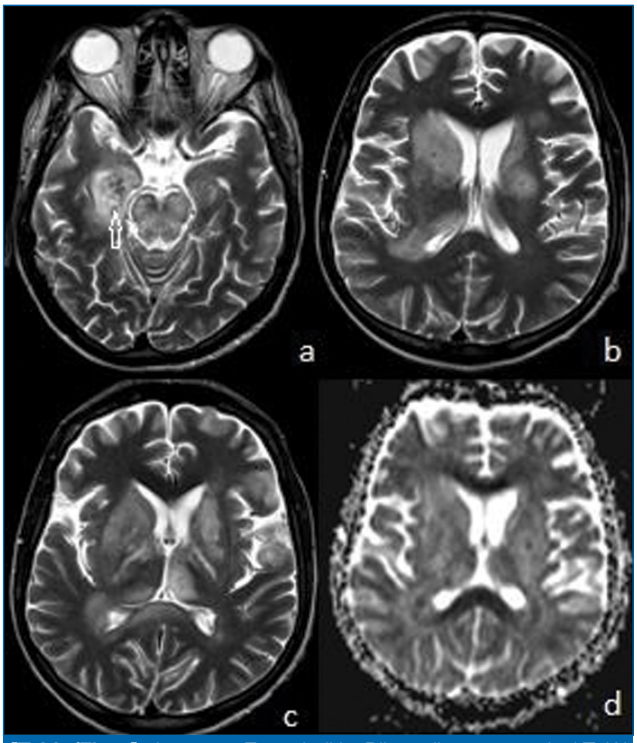


[Table/Fig-3]: Herpes Encephalitis: Axial T2W images show hyperintense signal involving cortex and underlying white matter in right temporal lobe, hippocampus, basifrontal region, cingulate gyrus and insular cortex (a,b). Foci of blooming are seen in the involved area on GRE image (black arrow, c) which also shows reduced ADC values (d).

One of the patients with herpes encephalitis in follow up after four months clinically presented with Kluver Bucy Syndrome and revealed marked atrophy of the temporal lobes on MRI. Atrophy of hippocampus and parahippocampal gyrus as sequelae to herpes encephalitis is a well established fact [5].

Unilateral basal ganglia and thalamic involvement is less common and was seen in three patients [6]. A patient of herpes encephalitis presented with fever and altered sensorium and his MRI scan was normal, but CSF revealed lymphocytosis. This patient responded clinically to acyclovir. Normal imaging has been reported in herpes infection [7]. Diffusion restriction was observed in 5 patients, which indicate fulminant clinical course. One of them died of marked cerebral oedema and transtentorial herniation. Sener RN, in their study opined that such sequelae is due to cytotoxic oedema and causes sudden clinical deterioration [8]. The diagnosis of HSV encephalitis has been greatly enhanced by the development of Polymerase Chain Reaction (PCR) analysis of CSF, which can detect all major herpes viruses that cause neurologic disease [9].

In patients with Japanese encephalitis, abnormal T2/FLAIR hyperintense signal with diffusion restriction was noted in the thalami, basal ganglia and the mid brain bilaterally. In one case, there was further involvement of the pons and medulla.



[Table/Fig-4]: Japanese Encephalitis: Bilaterally asymmetrical T2W hyperintense signal seen in brainstem, medial temporal lobes, basal ganglia, thalami, internal capsules and peritrigonal white matter with markedly hypointense foci suggestive of haemorrhage (a, white arrow). Foci of diffusion restriction are seen in left thalamus and bilateral basal ganglia (d).

Thalamic haemorrhages were seen in seven patients. Findings of diffusion restriction in deep grey matter nuclei and brainstem with haemorrhage in thalami were quite specific to Japanese encephalitis [Table/Fig-4].

Similar to our results, Kumar S et al., documented bilateral thalamic haemorrhages in 71% patients of Japanese encephalitis. Haemorrhagic lesions of brainstem and cerebellum have also been described [10].

In the present diffusion restriction was seen in all cases of Japanese encephalitis. This is almost always seen in Japanese encephalitis and helps in the characterisation of the duration of the lesions [11].

Acute lesions of viral encephalitis may mimic neoplasm especially if unilateral and are showing diffusion restriction. Short history, fever, MRI findings and response to antiviral drugs and steroids point towards the diagnosis of encephalitis. Imaging changes must always be evaluated in conjunction with the clinical presentation and laboratory abnormalities, especially the presence of CSF pleocytosis [12].

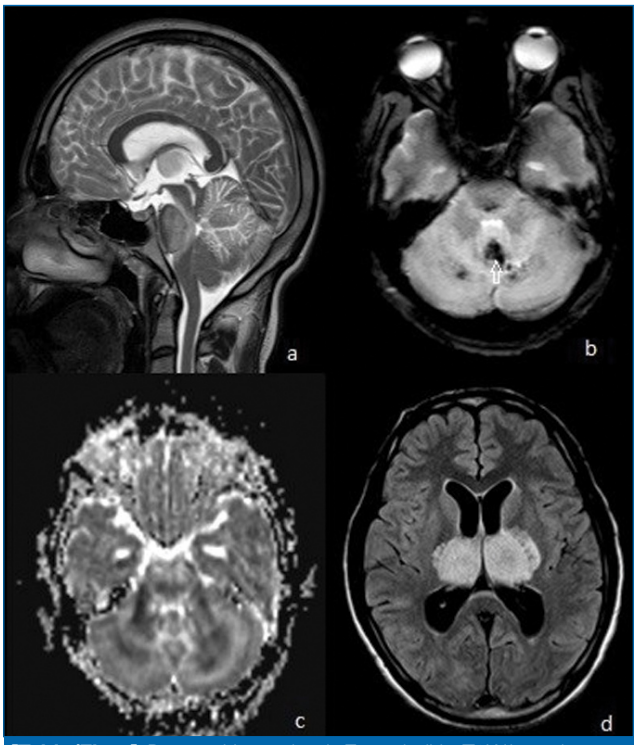
There is limited role of advanced MRI techniques like MR Spectroscopy (MRS) in the differential diagnosis of encephalitis. It has been described that encephalitis may result in reactive proliferation of cellular elements of the glial tissues and immune system, producing MRS profiles similar to gliomas. MR spectroscopy in encephalitis generally shows decreased NAA/Cr ratios at various time intervals, due to neuronal loss and an increased Cho/Cr ratio due to myelin breakdown [13]. Increase in NAA/Cho ratio suggests recovery.

Four patients were hospitalised with altered sensorium, fever and thrombocytopenia. They were seropositive for dengue antigen. On MRI, they showed bilateral thalamic involvement. Diffusion restriction and haemorrhage were seen in two of them [Table/Fig-5]. Leptomeningeal enhancement and cerebellar involvement were seen in one patient each.

Bhoi SK et al., demonstrated thalamic and basal ganglia lesions, meningeal enhancement and multifocal cortical and white matter lesions in dengue encephalitis. They also opined that cerebral imaging features was not related to hematological and biochemical changes or outcome. Our patients had complete clinical recovery and were discharged [14].

Two patients presented in altered sensorium following dog bite. Vaccination following the bite was reported in one case only. On MRI, both had T2/FLAIR hyperintensities in the brainstem, thalami, hippocampi and hypothalamus. Basal ganglia and cervical myelitis were also seen in one case each. Diffusion restriction and haemorrhage were absent. Both of them died within days due to respiratory complications [15].

T2 hyperintensity in the hypothalamus was found to be a constant feature of rabies encephalitis and absence of



[Table/Fig-5]: Dengue Hemorrhagic Encephalitis: T2W hyperintense signal abnormalities in cerebellum and brainstem (a,b,c) thalami and posterior limbs of internal capsules (d) which show haemorrhage (white arrow,b) and patchy diffusion restriction (c).

diffusion helped to differentiate rabies encephalitis from other viral rhombencephalities or Japanese encephalitis [16].

One of the patients presented with weakness of both lower limbs. MRI showed abnormal T2W hyperintense signal without diffusion restriction in the brainstem, cerebellum and the cervicomedullary junction. CSF showed pleocytosis (200 cells) and the diagnosis of encephalitis was suggested. The patient responded dramatically to steroids and improved. Hence, the final diagnosis of post infectious demyelination (ADEM) was considered. Similar imaging manifestations were also seen in other cases of ADEM, who include changes in the cerebellum (post viral cerebellitis), long segment hyperintense signal in the cord (post viral myelitis) and in the basal ganglia. Diffusion restriction was noted in few of them. Past history of fever was present in all the cases. ADEM/Post infectious encephalomyelitis, usually occurs after vaccination in past four to six weeks or an infection which may be a childhood exanthema like measles, rubella or chickenpox, or systemic infection affecting the respiratory or gastrointestinal systems [17].

In ADEM, lesions can be seen anywhere in the cerebrum-cortex, white matter, basal ganglia and brainstem.

Multifocal, asymmetrical areas of demyelination which are few in number and mostly non haemorrhagic located in the brain

stem, cerebrum, and cerebellum are considered characteristic of ADEM. Such lesions also correlate with clinical symptoms and signs [18].

A 6 days old male child with normal birth history presented with abnormal body movements. Neuroimaging revealed bilateral punctate foci of T1 hyperintensity with diffusion restriction. No meningeal enhancement or hydrocephalous was seen. CSF revealed only two cells. The diagnosis of viral encephalitis was considered by excluding other differentials. The patient responded to antivirals and supportive therapy. Similar spectrum of imaging has been described in parechovirus infection [19].

Children with Cytomegalovirus infection revealed malformations like microcephaly with polymicrogyria or delayed development with pachy/polymicrogyria on imaging. All of them had positive IgG antibodies for Cytomegalovirus.

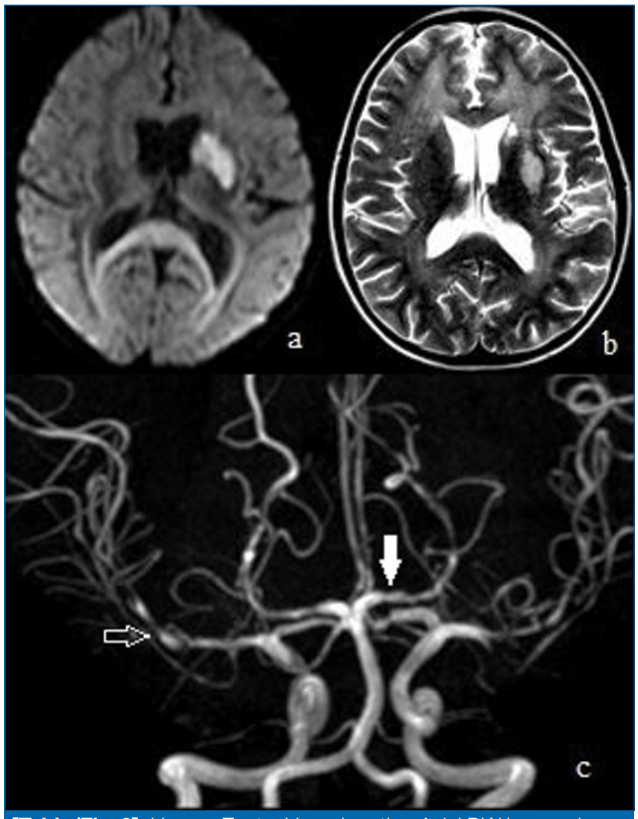
Cerebral malformations in congenital cytomegalovirus infection have been documented. Lissencephaly, cerebellar hypoplasia, calcifications and pachy/polymicrogyria have been reported in proven cases of congenital cytomegalovirus infection [20]. Anterior temporal lobe cysts involving the subcortical white matter and parenchymal calcifications especially in periventricular location are also features of congenital CMV infection [21].

It has been suggested that in children with cortical malformations, congenital Cytomegalovirus infection should be ruled out [22].

Nine patients with recent history of herpes zoster and clinical symptoms, underwent MRI. Neuroimaging revealed haemorrhagic white matter lesions and were labeled as hemorrhagic herpetic leukoencephalitis. Varicella zoster is known to cause classical acute haemorrhagic, necrotising encephalitis with innumerable white matter lesions in cerebrum [23].

A HIV positive child presented with repeated attacks of shingles and right sided weakness. Neuroimaging revealed acute infarct in the left basal ganglia. Concurrently bilaterally symmetrical frontal white matter hyperintensities in the subcortical and deep white matter were seen. The white matter hyperintensities were attributed to HIV encephalitis. Patient was started on HAART. Three weeks later, he presented with weakness in the right leg and had acute infarct in left MCA territory. MR angiogram revealed irregular narrowing of large and medium blood vessels of MCA and PCA bilaterally. The diagnosis of herpes induced vasculopathy and infarction was entertained [Table/Fig-6].

Similarly Picard O et al., demonstrated hemispheric stroke due to segmental arteritis of the carotid siphon in HIV positive patients who had no clinical history of zoster dermatitis. Varicella zoster virus (VZV) was found in the CSF. It is suggested that VZV must be considered in HIV positive patients presenting with hemiparesis [24].



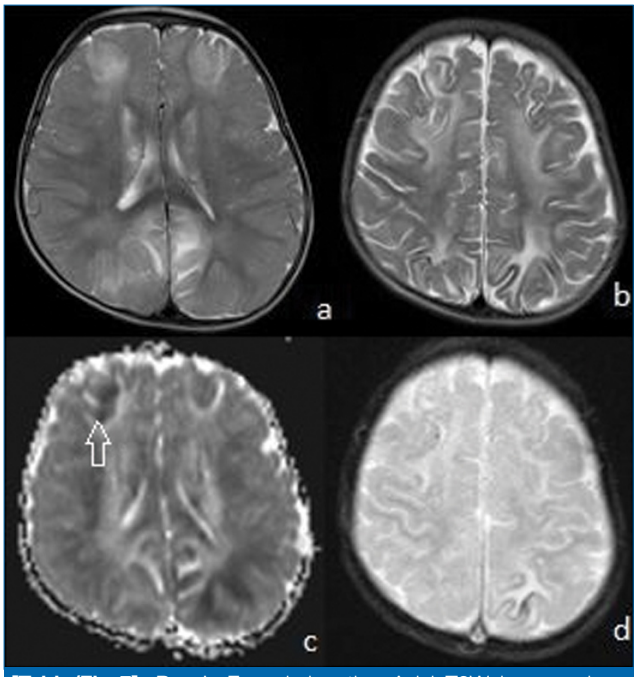
[Table/Fig-6]: Herpes Zoster Vasculopathy: Axial DW image shows diffusion restriction suggestive of acute infarct in left lentiform nucleus (a). Altered T2W hyperintense signal is seen in bilateral white matter suggestive of encephalitis (b). Contrast enhanced MR angiography shows narrowing of distal M1 segment of left MCA right MCA (white arrow) and beaded appearance of bilateral PCA (solid arrow)- Suggestive of medium vessel involvement.

VZV vasculopathy in the brain affects large and small vessels. It is opined that immunocompetent individuals usually have large-vessel disease and small vessel involvement is commoner in immune-compromised patients. In some patients, large and small vessel involvement can be found simultaneously [25].

One patient presented with right 7th and 8th nerve palsy with facial rash, MRI revealed unilateral cerebellar atrophy. The patient was later clinically diagnosed as Ramsay hunt syndrome (Herpes oticus) and responded to the combination treatment of oral acyclovir and corticosteroids. Herpes Zoster can cause granulomatous angitis in the brainstem or cerebellum and cause focal neurological deficit [26].

Non-specific viral encephalitis was diagnosed in seven patients and no virus could be pin-pointed as aetiological agent. Due to overlapping imaging findings and atypical presentations, specific viral agent could not be determined in these cases [27]. However, one must consider that herpes, dengue and Japanese encephalitis form a major group of viral encephalitis and are more common in our region. So recognition of neuroimaging patterns of these viruses as aetiological agents can solve the diagnostic dilemma in most of the patients.

A 10-year-old boy with history of blood transfusion presented with fever. MRI showed bilaterally symmetrical T2W/FLAIR hyperintense signal in the watershed territory cortex and subcortical white matter [Table/Fig-7] sparing the basal ganglia and thalami. Imaging differentials were viral encephalitis and ADEM. Altered liver function tests and aspirin intake pointed towards the diagnosis of Reye's syndrome. Involvement of subcortical white matter was observed in Reye's syndrome in our study. Signal alterations and diffuse cerebral oedema with diffusion restriction in brainstem, thalami, subcortical white matter, cerebellum, medial temporal lobes and parasaggital cortex have been reported in Reye's syndrome in the literature [28].



[Table/Fig-7]: Reye's Encephalopathy: Axial T2W images show hyperintense signal in bilateral subcortical white matter (a,b) which show patchy diffusion restriction (white arrow, c) and blooming suggestive of haemorrhage on GRE (d).

Clinical Significance

The present study highlights that clinical presentation and neurological signs of various viral encephalitis and ADEM overlap. MRI is of utmost importance in reducing mortality from these diseases as it delineates the anatomical sites of involvement and imaging characteristics specific to certain viruses like diffusion restriction, contrast enhancement and hemorrhagic lesions which give a clue to the radiologist and clinician about the aetiological agent. This study shows common viral encephalitis prevalent in this region of world and their classical MRI features, so as to guide the clinicians in early diagnosis and patient management.

LIMITATION

The major limitation of this study was small sample size especially if the data analysis was split by aaetiology. Secondly, the diagnostic accuracy of MRI was influenced by the clinical history. Except in the classical cases of Herpes and Japanese encephalitis, the accuracy with ADEM and certain rare encephalitis is very low in the absence of clinical profile. Further research is needed to better define the diagnostic criteria especially in less common types of encephalitis.

CONCLUSION

Most common presentation of viral encephalitis and ADEM is fever and altered sensorium. Asymmetric altered signal intensities in medial temporal lobes, cingulate gyri and insular cortices are characteristic of herpes simplex encephalitis. Thalamic haemorrhages with/without basal ganglia and brainstem involvement point towards dengue or Japanese encephalitis. Diffusion restriction and haemorrhages are uncommon in the brainstem lesions of rabies. Cortical malformations may be a part of congenital CMV encephalitis. Varicella zoster vasculopathy can involve both large and small vessels and must be considered in immunosuppressed patients presenting with recurrent strokes. Diagnostic challenge between ADEM and viral encephalitis may be solved with empirical treatment with steroids in patients with history of fever. Thus, we conclude that MRI is the modality of choice for diagnosing viral encephalitis and suggesting specific virus in an appropriate clinical and biochemical setting.

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Note: A part of manuscript has been presented before in a previous study of ours [29].

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