Imaging of infective causes of childhood epilepsy

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Abstract:

Introduction: A seizure or convulsion is a paroxysmal, time limited change in motor activity and/or behaviour that results from abnormal electrical activity in the brain. Epilepsy is a chronic condition characterized by recurrent seizures unprovoked by an acute systemic or neurological insult. Radiological evaluation of seizures done mainly by computed tomography (CT) & magnetic resonance imaging (MRI). MRI has been shown to be superior to CT for the detection of cerebral lesions associated with epilepsy. The advantage of CT lies in its wider availability, lower costs, less time (helpful in emergency) and compatibility with metallic implants in the patient.

Aim : of the study was to evaluate causes, to elucidate infective causes of epilepsy by CT and MR Imaging, to characteristics of the different infective causes.

Materials & methods: It was an observational study on 20 patients carried out in the Department of Radiodiagnosis, Silchar Medical College & Hospital, Silchar from August 2014 to July 2015 under 15 years of age using CT and MRI .

Results: Adoloscents constituted 6 (30 %) of the total 20 patients, most are males 15 (75 %). Inflammatory granuloma constituted 10 (50%) of the cases, HSV encephalitis 3 (15 %) , Japanese encephalitis 2 (10 %).

Active form of NCC constituted the predominant granulomas (50 %), followed by tuberculoma (30 %) and calcified NCC (20%) on MRI. Solitary granuloma constitute 60% of patients , followed by multiple (40%). Mild ventriculomegaly constituted commonest feature of pyogenic meningitis

Conclusion: Radiological imaging in the form of CT and MRI play a vital role in etiological evaluation in cases of epilepsy. MRI is the first choice in case of epilepsy. MRI is also recommended, as it is a radiation free modality, however CT is superior in identifying calcified lesions. Infections constitutes one of the important cause of epilepsy .

Keywords: Epilepsy, infective, CT, MRI.
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Introduction:

A seizure or convulsion is a paroxysmal, time limited change in motor activity and/or behaviour that results from abnormal electrical activity in the brain.[1]

A post-ictal period of decreased responsiveness usually follows most

seizures, in which the duration of the post-ictal period is proportional to the duration of seizure activity.[2] Seizures can cause involuntary changes in body movement or function, sensation, awareness, or behaviour.

Epilepsy is a chronic condition characterized by recurrent seizures unprovoked by an acute systemic or neurological insult. An epileptic seizure is a clinical manifestation of abnormal, excessive neuronal activity arising in the grey matter of the cerebral cortex.[3]

Radiological evaluation of seizures done mainly by computed tomography (CT) & magnetic resonance imaging (MRI).

MR imaging has emerged as the more diagnostically valuable and most valuable tool because of its 1) excellent soft tissue contrast, allowing for detailed depiction of anatomy, 2) freedom from beam-hardening artefact in basal brain that occur with CT and 3) capacity for multi-planner imaging.

MRI has been shown to be superior to CT for the detection of cerebral lesions associated with epilepsy.It scores over CT for better lesion depiction & characterization, better contrast resolution, better grey white matter differentiation and lack of ionizing radiation. The use of newer MRI methods, such as Fluid Attenuated Inversion Recovery (FLAIR), has increased the sensitivity for detection of abnormalities of cortical architecture.

The advantage of CT lies in its wider availability, lower costs, less time (helpful in emergency) and compatibility with metallic implants in the patient.

Aim of the study was to evaluate the infective causes of epilepsy, to elucidate infective causes of epilepsy by CT and MR Imaging, to study the CT and MR imaging characteristics of the different infective causes.

Materials & methods:

The present study was carried out in the Department of Radiodiagnosis, Silchar Medical College & Hospital, Silchar from August 2014 to July 2015.Inclusion criteria: All patients under 15 yrs of age presenting with seizures with suspected infective cause with imaging revealing an infective lesion in the brain, were included in this study.

Exclusion criteria: Those patients who were contraindicated for CT examination and claustrophobic patients were excluded from the study. Informed consent was obtained from the subjects before the commencement of the investigation. In all cases a thorough history was taken including treatment history. Brief general examination was done in all cases. Routine blood test reports were collected, EEG report noted if available. Before subjecting the patient to MRI, special precaution was taken about metallic implants, pacemaker.

Technique:

CT evaluation was carried out using a SIEMENS SOMATOM EMO (2003).

CT protocol:- Factors of 130 KV and 70 MA were a constant feature of all cases. Routine axial scans were performed in all the cases taking orbitomeatal line as the base line. 5 mm slice thickness for the posterior fossa and 8 mm slice thickness for the supratentorial region were employed routinely. Thin contiguous slices of 2 mm or 3 mm were taken wherever necessary. Initial NECT scans were performed in all cases and in specific indications CECT scans were taken after intravenous bolus administration of 50 ml of non-ionic contrast media. The patients were asked to have nothing orally for at least 4 hours preceding the scan, in case intravenous contrast was needed. Images were interpreted in the cerebral & bone window and manipulated according to need.

MRI evaluation

MRI protocol:- The scenario and length of the examination were explained in understandable terms to the parents and patients. Sedation was used as and when required with profofol infusion 50-100µg titrated to desired level of sedation. Anxiolytic medications were used in claustrophobic patients. Patients were positioned supine, head first, placing the

head properly in head coil. The patients were positioned as comfortably as possible to minimize movement artifacts. Scanning was done in the axial, coronal and sagittal planes. Sequences used are T1W, T2W, Fluid attenuated inversion recovery (FLAIR), GRE in all patients and Proton density (PD), Post Gadolinium T1W, susceptibility weighted imaging (SWI), Diffusion weighted imaging (DWI), Apparent diffusion coefficient mapping (ADC Map), Multivoxel MR Proton Spectroscopy (MRS), 3D SPoiled Gradient Recalled (SPGR) sequence, MR angiography and venography in selected cases.

MR parameters: T1W FSE: TR=475ms, TE=7.8ms, Bandwidth=130, Slice thickness= 5mm, FOV= 201x230, Flip angle=90degrees.

T2W FSE: TR=4000ms, TE=94ms, Bandwidth=130, Slice thickness=5mm, FOV=201x230, Flip angle=150degrees

Proton density used as an alternative to FLAIR, and is more sensitive for the detection of posterior fossa lesions. TR=2600ms, TE=27ms, Slice=5mm, FOV=201x230, Flip angle=90degrees

FLAIR + Gd -- Indicated for the detection of leptomeningeal disease

TR=9000ms, TE=90ms, TI=2500ms, Slice thickness=5mm, FOV=201x230, Flip angle=150degrees.

DWI /ADC TR=2700ms, TE=88ms, BW 1500, Slice thickness=5mm, FOV=230 x 230, Flip angle=90 degrees.

T2* GRADIENT TR= 729, TE= 26, BW= 80, flip angle= 20 degrees, FOV= 201 X 230, slice thickness =5 mm,

SWITR=49ms, TE=40ms, FOV=201 x 230, Slice thickness= 2 mm, Flip angle=15degrees.

MPGRTR=1920ms, TE=2.7 ms, TI=1100ms, FOV=192X256, BW= 280, Slice thickness=0.90mm, Flip angle=15 degrees.

T2 RELAXOMETRY for hippocampal sclerosis

TE1=22ms to TE16=352ms, TR=3000ms, FOV=168X256, Slice thickness = 5mm, Flip angle=180 degrees.

MULTI VOXEL MRSTR=1690ms, TE=135 ms, Flip angle=90 degrees.

Results and observations:

The study included 20 patients under 15 yrs of age, with recurrent seizures referred from the department of Medicine, Psychiatry & Paediatrics of Silchar Medical College & Hospital, Silchar. Cases with at least two attacks of unprovoked seizures were included in the study.

Those patients with seizures, undergoing CT and/or MRI study, but with normal study or other than infective causes, were not included in this study.

Those patients with immediate post-traumatic seizures were also not included in this study, as accurate history of the episode of seizure could often not be taken in these patients.

The patients with post-ictal transient changes confirmed on follow up imaging were also excluded from the study.

The results and observations are as follows:

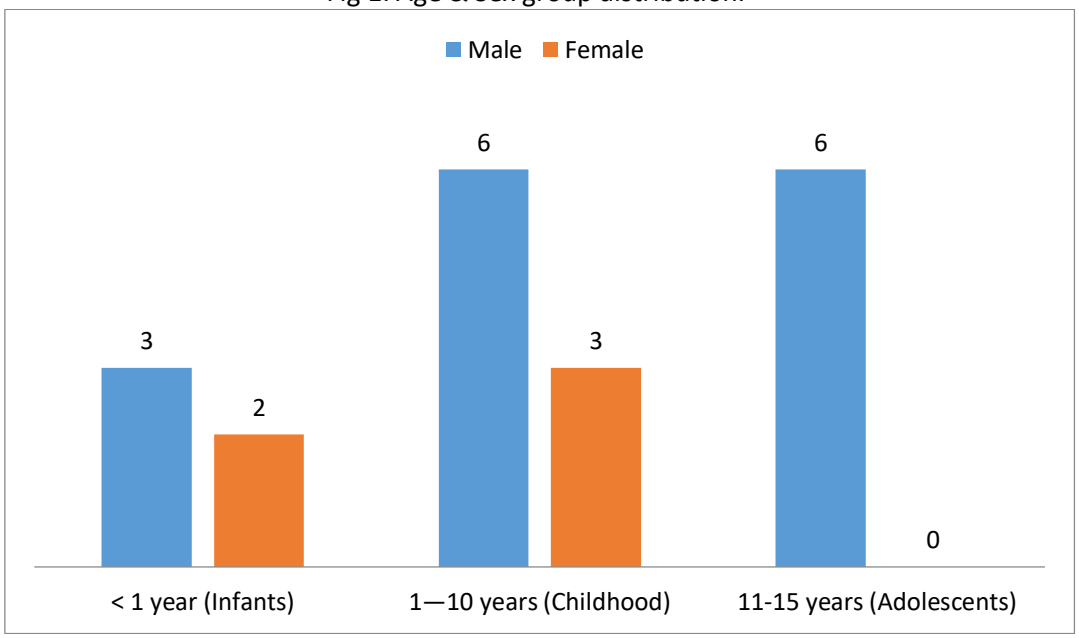
Table 1: Distribution of cases in various age groups

AGE GROUP	NUMBER (n)	PERCENTAGE (%)
< 1 years(Infants)	5	25
1—5 years (Early Childhood)	5	25

6-10 years (School aged children)	4	20
11-15years(Adolescents)	6	30

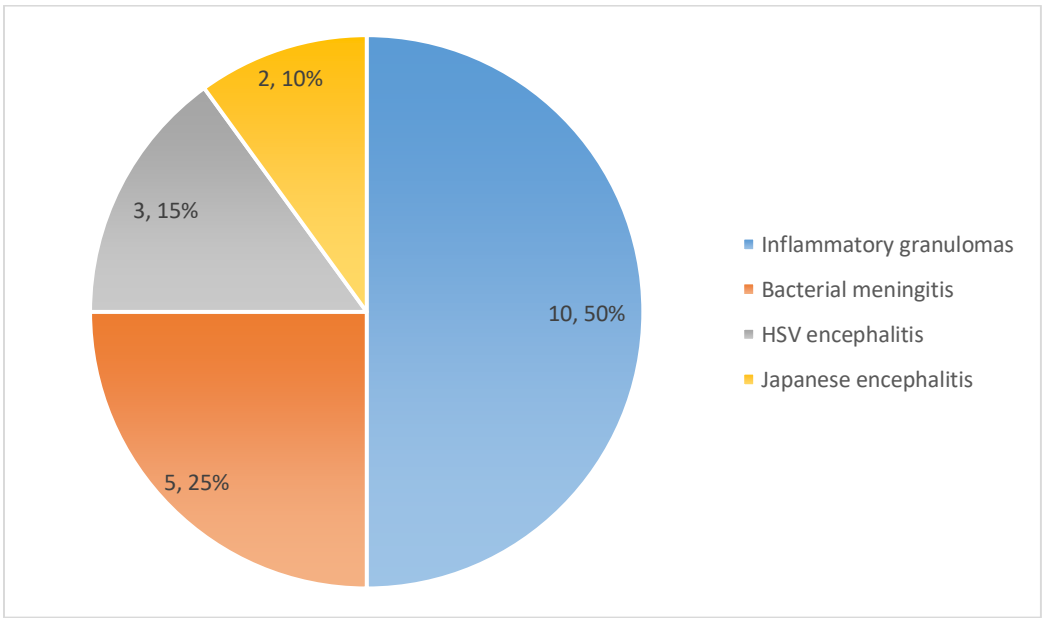
Adoloscents constituted 30 % of the total cases (Table 1).

Fig 1: Age & Sex group distribution.



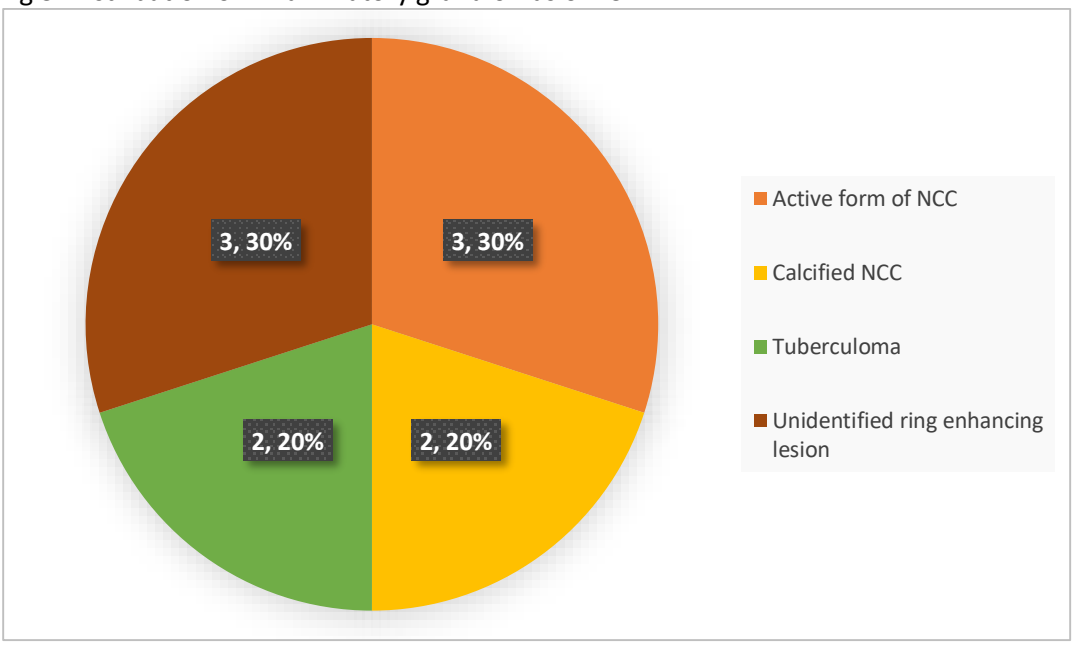
Males constituted 75 % of the total cases (Fig 1).

Fig 2: Distribution of infective etiology.



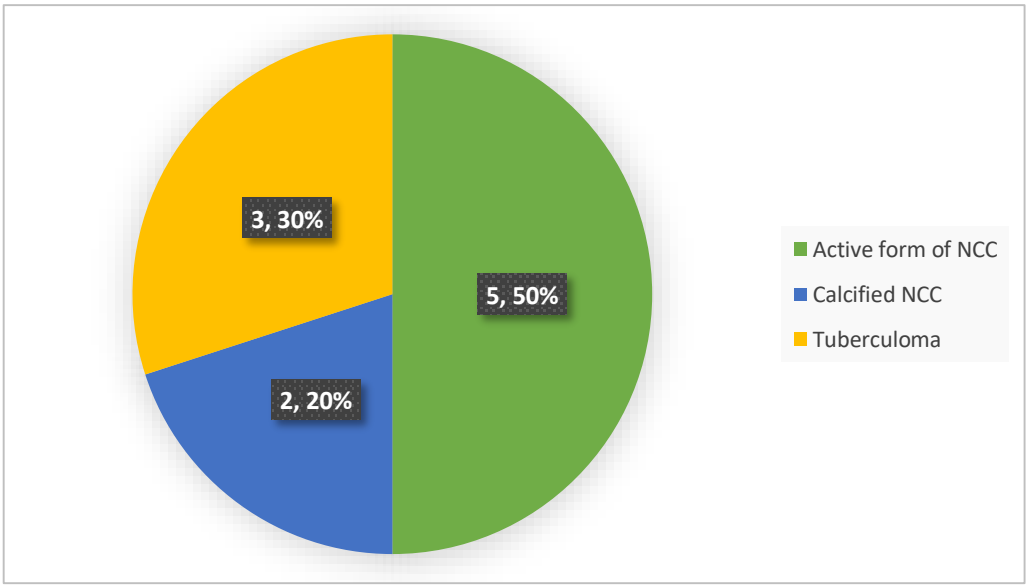
Infective granulomas constitutes 50% o cases (Fig 2)

Fig 3: Distribution of inflammatory granulomas on CT



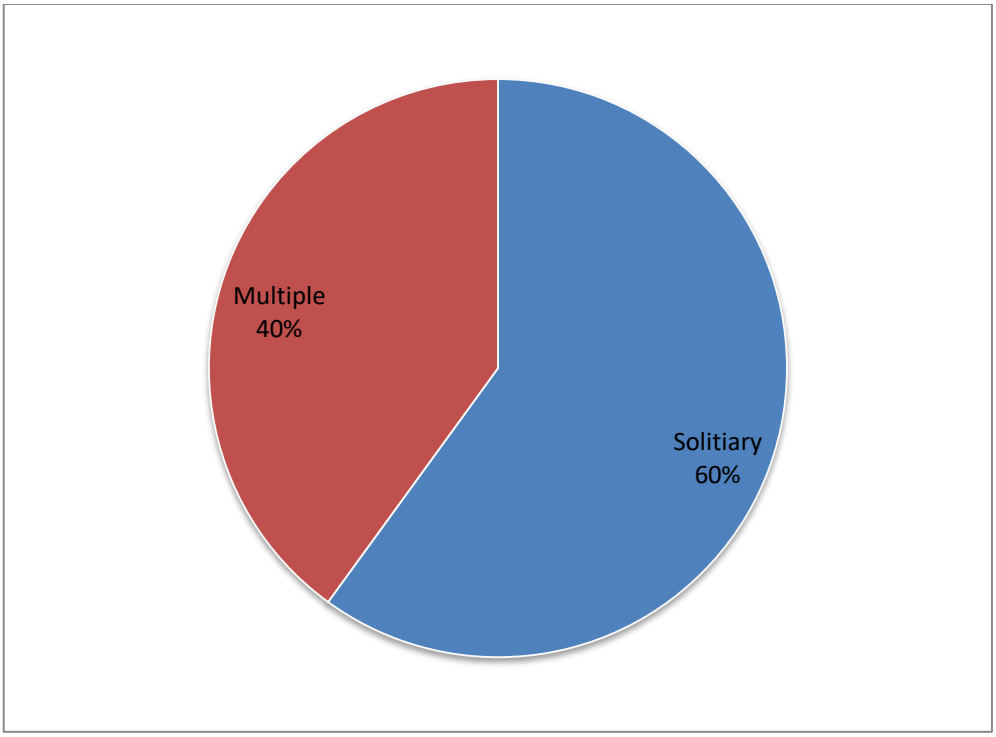
Active form of NCC (30%) constituted the predominant granulomas on CT (Fig 3).

Fig 4 : Distribution of inflammatory granulomas in MRI.



Among the inflammatory granulomas in MRI, active form of NCC forms 50% of cases (Fig 4).

Fig 5: Inflammatory granulomas- Solitiary vs Multiple.



Among the inflammatory granulomas, solitiary granulomas constitutes 60 % of cases (Fig 5).

Table 2: Stages of NCC.

Stage	No. of cases	% of cases of NCC
Vesicular stage only	0	0%
Transitional stage (Colloid Vesicular, Granular Nodular)	5	71.43%
Calcified Nodular	2	28.57%

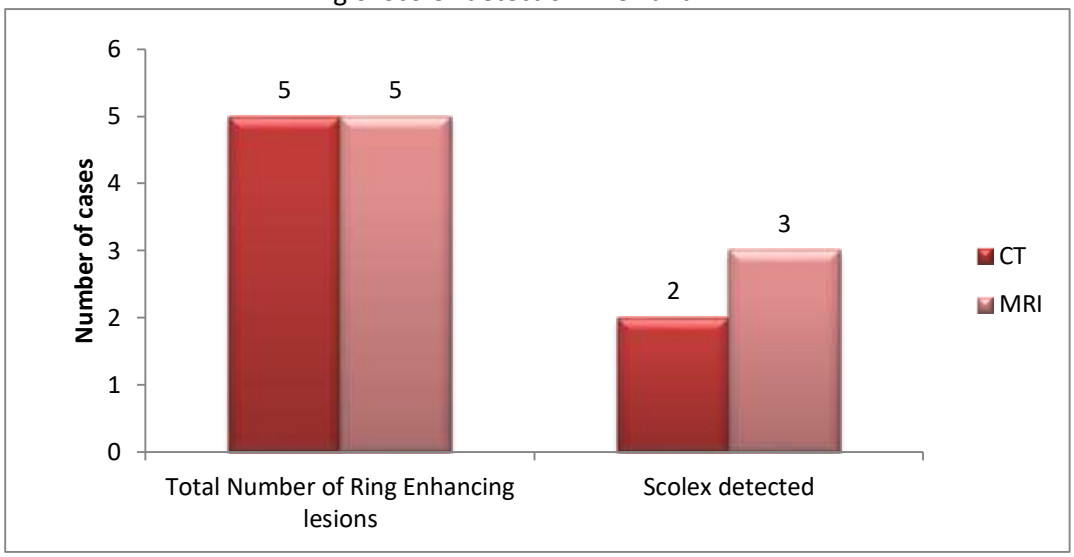
Transitional stages of NCC were predominant forms (71.43 %) (Table 2).

Table 3: NCC– Solitary v/s Multiple .

	Number of cases	% of NCC cases
Solitary	4	57.14 %
Multiple	3	42.86 %

Solitary form of NCC was more common than multiple form (Table 3) .

Fig 6: Scolex detection in CT and MRI.



CT detected 40% of the scolex detected on MRI (Fig 6).

Table 4: Degenerating NCC- pattern of enhancement (CT & MRI).

	RING ENHANCEMENT	DISC ENHANCEMENT
CT+MRI	100% (N=5)	0% (N=0)

Table 5: MRI features of active NCC.

Feature	Number of active cases of NCC	% of total active NCC cases
Size 10-20mm	5	100
Oedema	5	100
T1 weighted images		
Isointense(not seen)	3	60
Isointense periphery with low signal centre	2	40
T2 weighted images		
Low signal periphery with high signal centre	4	80
Isointense/ high signal periphery with low signal centre	1	20
Gadolinium enhanced study		

Enhancing peripheral ring with central low signal	5	100
Enhancing scolex	3	100
Total number of active NCC	5	100

All the active cases of NCC were 10-20 mm in size, showed perilesional edema and rim enhancement on post contrast studies (Table 5).

Table 6: MRI features of Tuberculomas.

Feature	Number of cases
Size 10-20mm	3
Oedema	3
T1 weighted images	
Slightly hyperintense centre	1
Isointense to brain	2
T2 weighted images	
Central hyperintensity	3
Hypointense rim	3
Gadolinium enhanced study	
Single rim enhancement	1
Conglomerated rim enhancement	2
Total number of Tuberculomas	3

Table 7: HSV Encephalitis: MRI Characteristics.

Location	Hyperintensity on T1WI	Hyperintensity on T2WI	Contrast enhancement	Hemorrhage
Unilateral Temporal Lobe	3	3	2	0
Bilateral Temporal Lobe	1	1	1	0
Cingulate Gyrus	1	1	1	0

Gyrus Rectus	2	2	1	0
Unilateral Frontal Lobe	2	2	2	0
Bilateral Frontal Lobe	0	0	0	0
Internal capsule	2	2	2	0
Meningeal enhancement	N/A	N/A	4	N/A

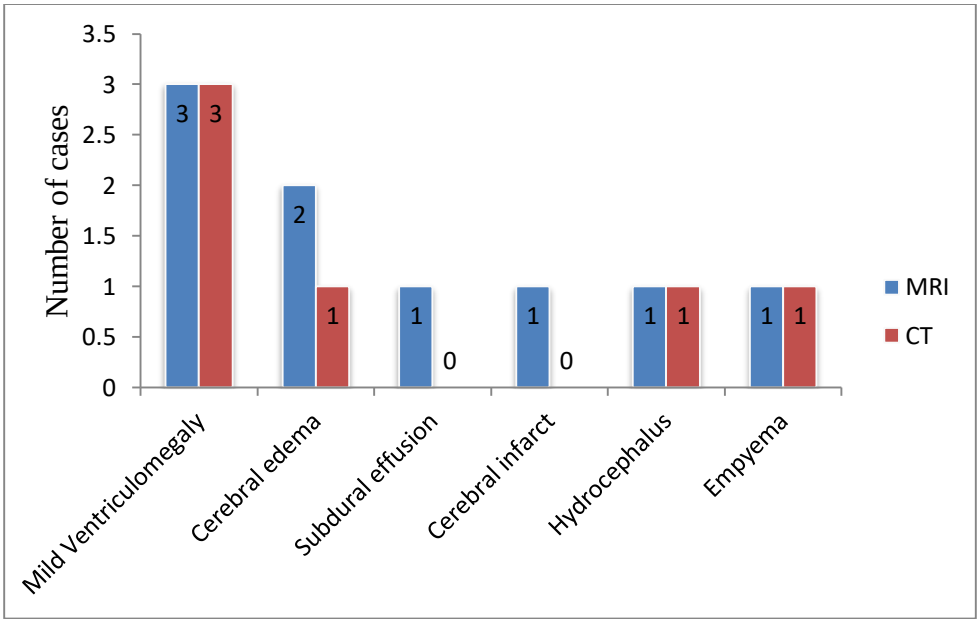
Table 8: HSV Encephalitis- CT Findings.

Findings	Number of findings
Diffuse edema	1
Temporal lobe involvement	4
Anterofrontal hypodensity	2
Frontoparietal hypodensity	0
Meningeal enhancement	4

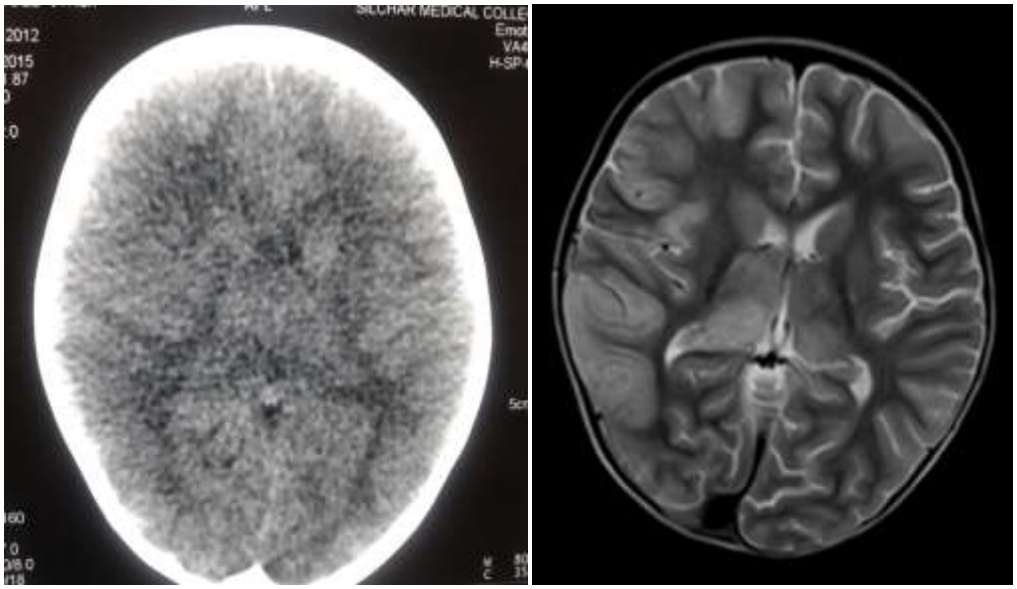
Table 9: Japanese Encephalitis- MRI Findings.

Region	T2 hyperintensity
Thalamus	2
Basal ganglia	1
Brainstem	1
Cortex	1

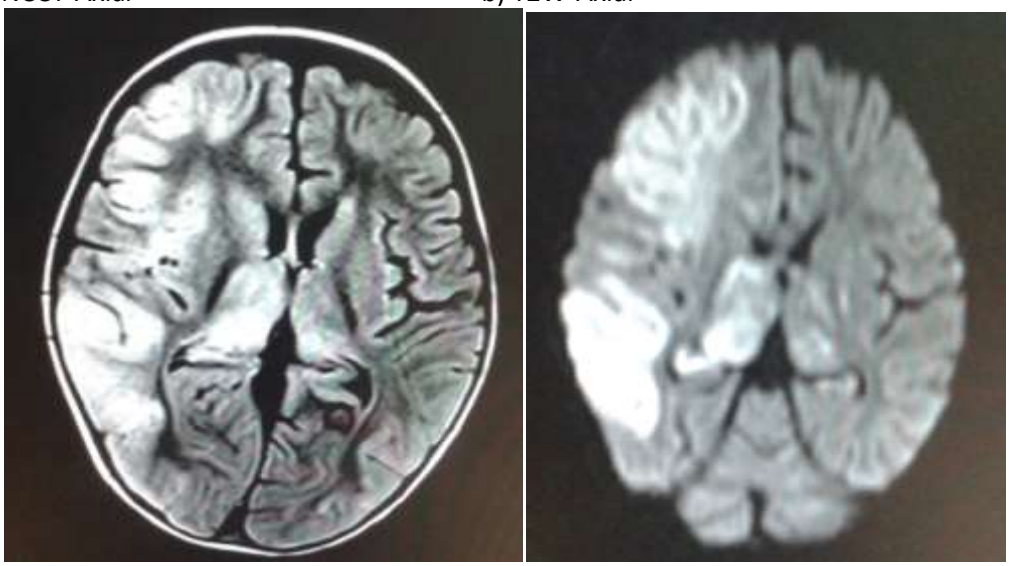
Fig 7 : CT and MRI features in Pyogenic meningitis.



Most common abnormality detected in cases of pyogenic meningitis was mild ventriculomegaly.

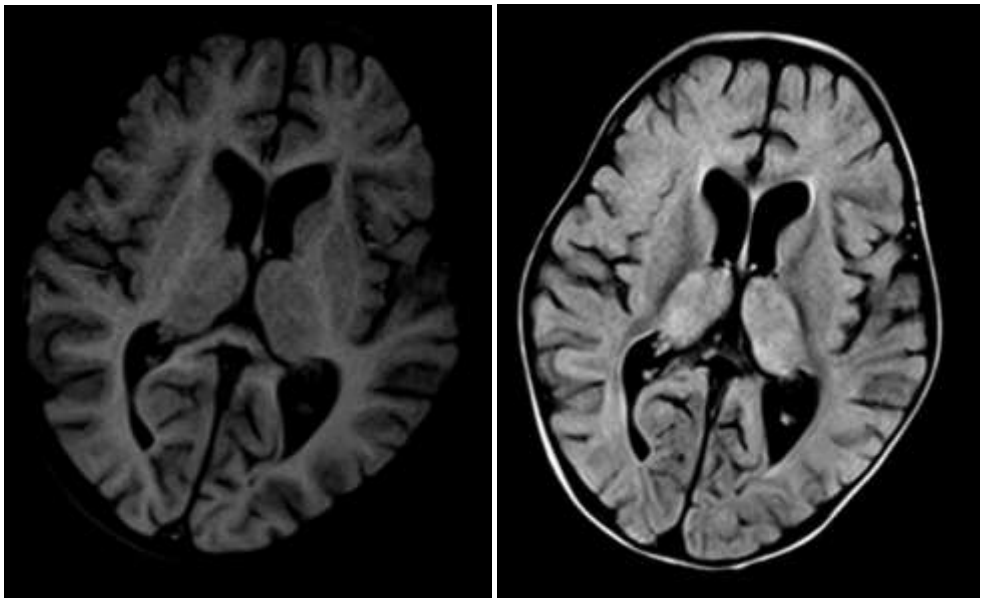


a) NCCT-Axial b) T2W-Axial

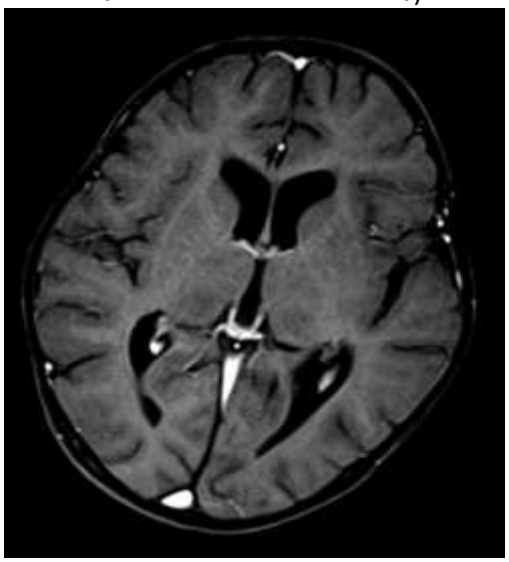


b) FLAIR-Axial d) DWI

Fig 8: HSV encephalitis : 3yrs old female patient presents with sudden onset of high grade fever, altered sensorium, and recurrent seizures. Diffuse edema noted on CT image. T2 and FLAIR hyperintensitynotedinvolving right frontal, parietal, temporal cortex and subcorticalwhite matter, right parahippocampalgyrus, insularcortex, bilateral thalami, right half ofpons, right cerebral peduncle, leftcoronaradiata, showingrestrictionon DW images, suggestive ofcytotoxicedema. HSV encephalitis.

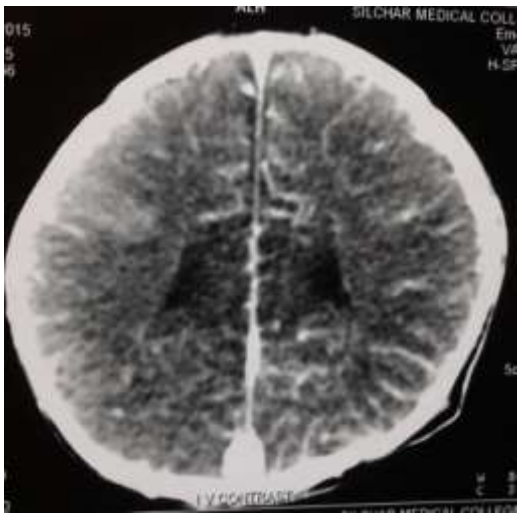


a) T1W-Axial b) FLAIR-Axial

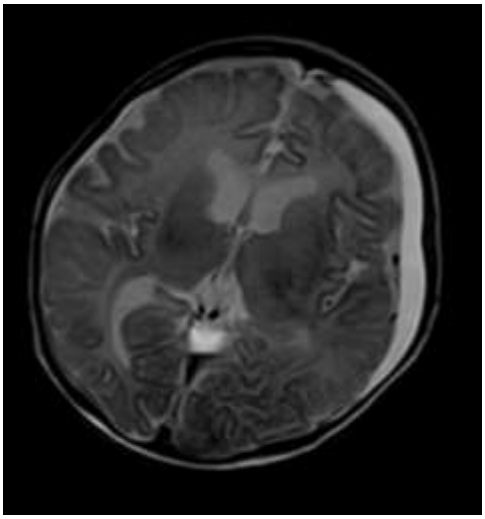


c) T1W-post Gd.

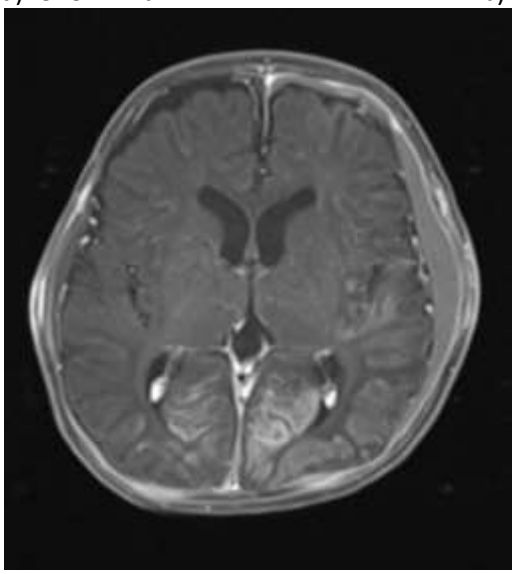
Fig 9: Japanese encephalitis: 5 yrs male presenting with altered sensorium, fever and recurrent seizures. T1 hypointensity, T2 hyperintensity noted in bilateral thalami, showing no post contrast enhancement.



a) CECT-Axial

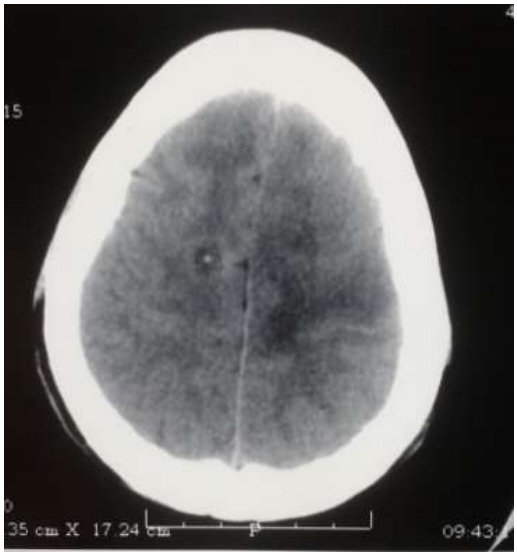


b) T2W-Axial

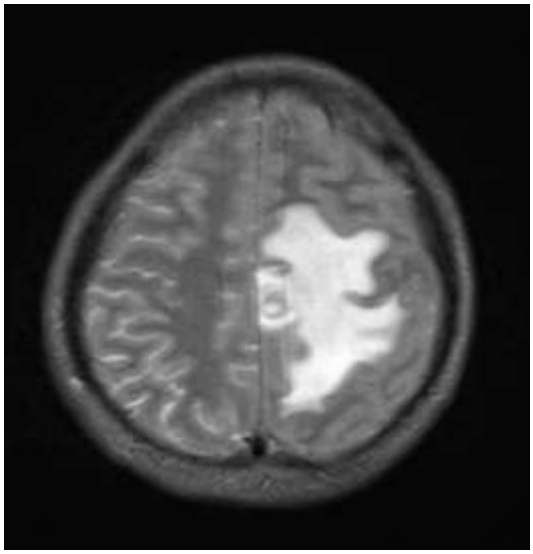


c) T1W-Post Gd.

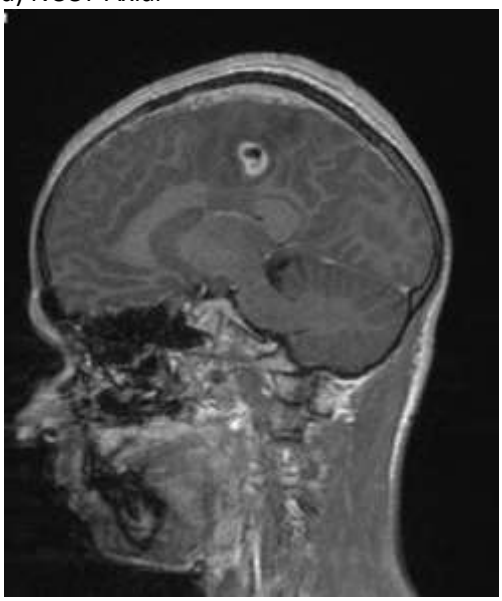
Fig 10: Pyogenic meningitis:3 yrs female presenting with altered sensorium, fever and recurrent seizures. Post contrast CT showing abnormal meningeal enhancement in left parietal region. T2 hyperintense extra-axial collection noted in left fronto-parietal convexity. Post Gd. T1W image showing gyriform enhancement in left parieto-occipital region. Pyogenic meningitis with subdural empyema.



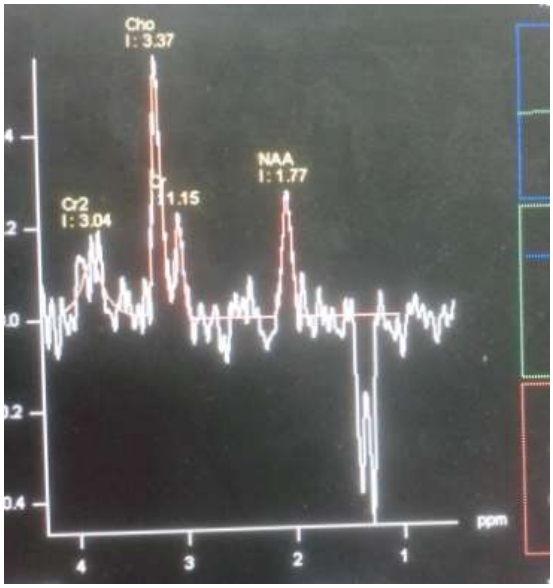
a) NCCT-Axial



b) T2W-Axial

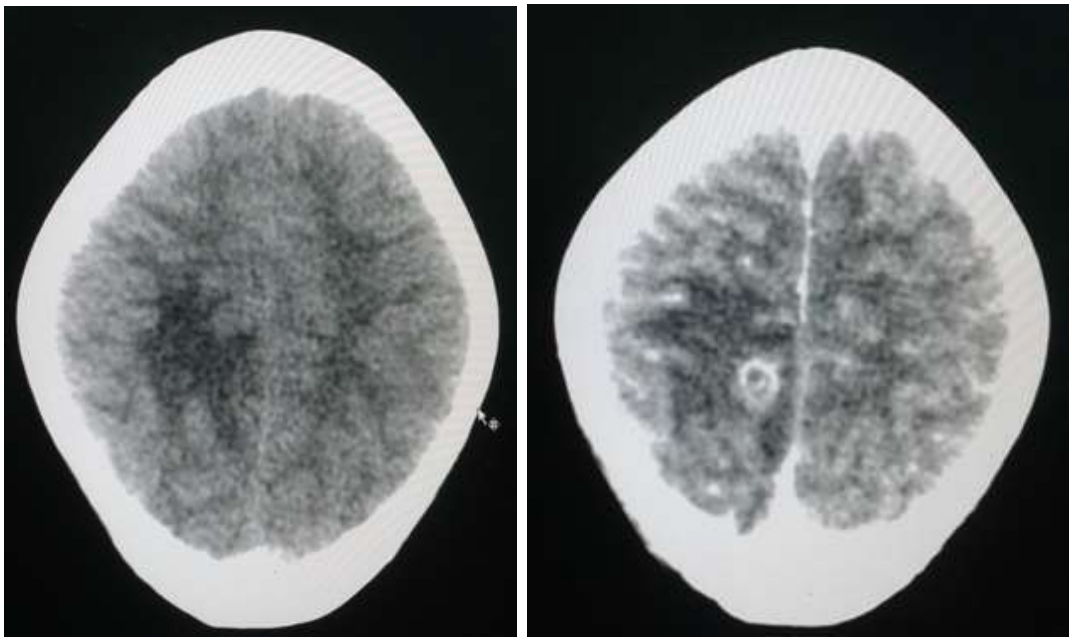


c) T1W-Post Gd.

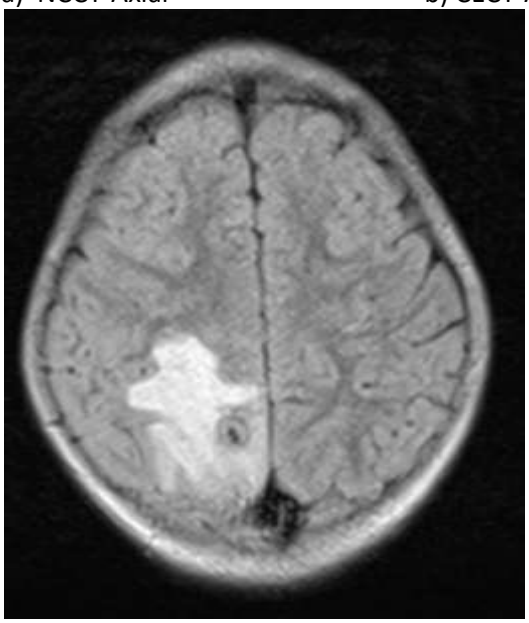


d) Spectroscopy

Fig 10: Tuberculoma:: 14 yrs male presenting with recurrent focal seizures. NCCT showing edema in left high parietal region. Ring artefact is seen in right high parietal region. A well defined T2 hypointense lesion with a T2 hyperintense centre noted in left high parietal region showing conglomerated rim enhancement. Lipid peak noted on spectroscopy. Tuberculoma.



a) NCCT-Axial b) CECT-Axial



c) GRE- Axial

Fig 11: NCC: 13 yrs male presenting with epilepsy. NCCT showing edema in right high parietal region. CECT showing rim enhancement and enhancing scolex. GRE image showing blooming artefact suggestive of calcification . NCC.

Discussion:

Our study was conducted in the Department of Radiodiagnosis, Silchar Medical College and Hospital, Silchar. We evaluated 20 cases of epilepsy of infective causes under 15 years of age for a period of one year using CT and MRI, the characteristics of which are discussed below.

Age distribution:

The whole group consisted of 20 patients. Age distribution was as

follows: 5infants (from 1 to 12 months of age), 5early childhood (from 1 to 5years), 4school aged children (6-10 years) and 6 adolescents (from 10 to 14 years). The maximum incidence of seizures secondary to an organic brain lesion was found in infants. Average age was 4.29 years, median 3 years.

Camfieldet al(1985)[4] in a population based study found an incidence of 118/1lakh person years for infants(<1years), 48/1lakh for those aged 1-5 years, 43/1lakh for those aged 6-10 years and 21/1lakh for those aged 11-15 years. The incidence of epilepsy was greatest in the first year of life, followed by a plateau between the ages of 1 and 10 years. After the age of 10 years, however, the incidence again decreased dramatically and approached that of adults.

HauserWA et al[5], Olafssonet al[6], Forsgren et al[7], Granieri et al[8], Sidenvall et al[9] in their studies concluded that age-specific incidence is consistently high in the youngest age groups, with highest incidence occurring during the first few months of life. Incidence falls dramatically after the first year of life, seems relatively stable through the first decade of life, and falls again during adolescence.

In our study, overall males predominate (61.5 %) (Fig 1) . Kwong et al[10](59%), Camfield et al[4], Shinnar et al[11](57%), Bharucha NE et al (68%)[12],Hauser WA et al[5], Olaffson et al[6] ,all show male dominance in incidence pattern.

The incidence of infectious etiology was found to be 27.2% of all the known etiology in a study by Bergamini et al[13] on 1785 epileptic childrens with CT scan.

Rim enhancing lesions

Mishra et al [182]in their study on 20 paediatric age group patients observed 15.7 % rim enhancing lesions.

In my study of the 20 cases studied on CT and MRI, 10 cases showed rim enhancing lesions. This comprised 20 % of total infective cases. All the cases were due to inflammatory granulomas. NCC alone formed 70% (N=7) of total rim enhancing lesions.

CT detected 3 out of 5 cases of NCC, and 2 out of 3 cases of tuberculoma (Fig 10). Thus 3 rim enhancing lesions remained unidentified with CT alone. On MRI examination of the 3 unidentified rim enhancing lesions, scolex was detected in 2 lesions suggestive of NCC. The remaining unidentified lesion was diagnosed to be tuberculoma on MRI scan with internal diffusion restriction and lipid peak on MR spectroscopy (Fig 10) .

Inflammatory granuloma :

The two types of inflammatory granulomas detected in this study were NCC and tuberculoma (Fig 10, Fig 11). Other types of inflammatory granulomas were not detected.

NCC made up 70% of this group and tuberculomas made up 30% (Fig 5) . 60% of the rim enhancing lesions were solitiary and 40 % multiple.

Hussain et al[14] in their study, out of total 94 granulomas, most common lesions were found to be solitiary ring enhancing lesions (80%), followed by single non- enhancing calcified granuloma (16%).

Neurocysticercosis:

In our study 7 cases of NCC were detected out of which 4 (57.14%) were detected in school aged children.

According to Garg et al[15] Kalra et al[16] and Singhi et al[22], school aged children are most commonly affected with slight male preponderance.

Cysticercosis is the most common parasitic infection of the CNS and seizure is its commonest manifestation.

In the present study, NCC comprised of 35 % of all infective cases. NCC as a cause of epilepsy has been reported to be 2.2-18.0 % in unselected cases of

epilepsy in India (Chopra J.S. et al[17]). Previous studyby Rajashekhar et al[18] show NCC granuloma constituted upto 48% of total neuroimaging positive cases in various community studies.

In our study it was observed that solitary NCC comprised 57 % of all the cases of NCC.

Wadia et al[19] studied 150 patients with simple partial seizures, and CT scanning revealed single enhancing lesions in approximately 26%.

According to Murthy et al[20], in a series of 558 children, 51% of symptomatic partial epilepsies were due to either active or inactive NCC.

According to Vykuntaraju et al[21], 44% (57/127) of the radiologically positive cases of paediatric epilepsy were due to neurocysticercosis.

In another study of 51 patients, Rajashekhar et al[18]documented cysticercal granulomas in 25 patients and tuberculoma in 6 ; in all cases these findings were confirmed. This gives a proportion of NCC: tuberculoma as 4:1. In the present study the proportion of NCC to tuberculoma was 2.3 :1.

In a study by Martinez HR et al[22]active forms including colloid vesicular and granular nodular constituted 85% cases of NCC.Inactive forms were observed better with CT. Singh G et al[23] also got incidence of 40 cases of such inactive lesions among 226 seizure cases (17.6%).

In this study, active form of NCC was predominant (71.4%) correlating with the study of HR Martinez et al[22]and Singh G et al[23].

Single small calcified granuloma at cortical subcortical location, with size <1 cm was taken as resolving calcified NCC as suggested by Singh G et al[23].

Revised diagnostic criteria for NCC consider that cystic lesions showing the scolex on CT or MR imaging are an absolute criterion for diagnosis of the disease, allowing its definitive diagnosis. (Del Brutto OH et al[24]).

Rajashekhar et al[18] in a study of patients of NCC, all the active cases showed perifocal edema. The commonest pattern was a low signal periphery with a high signal centre on T2 weighted images. The size of all the granuloma were 10-20 mm.

In this study all the active NCC cases showed perifocal edema. The most common pattern was found to be low signal periphery with a high signal centre on T2 weighted images (90%) correlating with the study of Rajashekhar et al[18].

According to Kishore D et al[25] in a study of 100 patients with NCC, majority of the cases had a ring like enhancement as seen in 88 out of 100 (88%) patients. Only 12% of the cases had disc like enhancement. An eccentric dot inside the ring representing the cysticercus larva was seen in only 19 out of 88 (21.6%) patients.

In this study 100% of active NCC cases showed ring like enhancement. Cases with multiple lesions mostly showed NCC lesions in various stages.

Garg et al[15]observed this eccentric dot (scolex) in 43% of patients.

In our study, scolex could be detected in 40 % on CT and 60 % on MRI of all the NCC cases.

In CT cases, my study correlates with the study by Garg et al[15].

The apparent high sensitivity of MRI over CT for detection of scolices may be due to multiplanar imaging in MRI, and as concluded by Bittencourt PR et al[26] , MRI often reveals more cysts than CT and offer more resolution to detect the scolex.CT could confidently detect 5 out of 7 cases of NCC detected by MRI.

In the study by Chamaria K et al[27], among the total of 565 lesions detected by both the tests, CT could detect 541 (95.7%) and MRI detected 561 (99.3%)

of lesions. The difference in the proportion of lesions detected between the tests was not significant (P value = 0.84,P > 0.05). Thus both MRI and CT were almost equally efficient in detecting parenchymal and intraventricular lesions in the brain.

According to Thakur et al[28], though the MRI is more sensitive, a contrast enhanced CT scan is probably good enough for transitional forms and is in fact superior for detection of calcifications.

According to a study by Rajashekhar et al[18], sensitivity of the imaging techniques: contrast-enhanced CT (5 and 10 mm slices) 93.8% (15/16); thin (2–5 mm) section contrast-enhanced CT 100% (10/10); Gd-enhanced MRI 100% (6/6); unenhanced MRI 93.8% (15/16). MRI did not reveal additional granulomas or cysts in any patient. In patients strongly suspected to be harbouring this lesion, when 10-mm contrast-enhanced CT reveals only oedema, thin (2–5 mm) slice CT is a cost-effective alternative to MRI.

Tuberculoma

The incidence of tuberculoma varies from 3 % to 24 % in different studies (Singhi et al[29] , Shorvon S[30]). The distinction between cysticercal granuloma & tuberculoma is controversial, often associated with single enhancing CT-documented lesions. This is because the clinical and imaging features are quite similar; both diseases are common in endemic areas and may coexist in the same patient.

Rajashekhar et al[18] have attempted to differentiate between the two entities on the basis of clinical and imaging features. Based on these findings & their experience, Rajshekhar& Chandy[31]in 1991 suggested that cysticerci are usually round in shape, 20mm or smaller in size, with ring enhancement or a visible scolex; cerebral edema severe enough to produce midline shift or focal neurological deficit is not seen. Tuberculomas in contrast are usually irregularly shaped, solid and greater than 20mm in size. They are often associated with severe perifocal edema and focal neurological deficit.

In this study Rajshekhar's criteria was used. However 2 cases in CT was diagnosed to be non specific as the lesion were 2 cm in diameter with well defined enhancing wall, minimal edema, no scolex. On MRI scan the lesions were diagnosed as tuberculomas. In MRI all cases could be characterised using the criteria of diffusion restriction and lipid peak on MR spectroscopy to diagnose tuberculosis (JayasundarR et al[32], Gupta et al[33]).

Lipids (in vivo proton spectra) as markers for tuberculoma were first suggested by Gupta et al[33].

In the study by Jayasundar R et al[32] lipid peak was observed in 86% of cases of tuberculoma. Lipid peak was not observed in 2 out of 28 tuberculoma lesions. The possible explanation for this was that the patients were subjected to MRI scans within 3 to 30 days post initiation of therapy. The author have suggested in a different study that responding tuberculomas exhibit progressive decrease in the level of lipids.

In the present study lipid peak was observed in 100% of cases of tuberculoma.

Meningoencephalitis:

In our study in infective group, 10 cases of inflammatory granulomas(50%) and 10 cases of meningoencephalitis(50%) were detected. 5 cases of bacterial meningitis, 3 cases of HSV encephalitis (Fig 8) and 2 cases of Japanese encephalitis (Fig 9) were detected.

According to a study by Davis et al[34] on 9 biopsy proven cases of HSV encephalitis with CT scan , a low density area in one

or both infero-medial temporal lobes(including part of the limbic system) with extension into the insular cortex, sparing the putamen, so that the lesions have a lateral convex or straight medial border is highly suggestive. The inferior frontal lobes, cingulate gyrus and thalamus may be involved.

In this study, involvement of inferomedial temporal lobes is observed in all the cases of HSV encephalitis with both CT scan and MRI correlating with the study of Davis et al[34].

According to Misra et al[35], on MRI, thalamus, basal ganglia and brainstem involvement are common and thalamic involvement has been reported to be suggestive of JE in an endemic area, especially in the post-monsoon period.

Thalamus was most commonly involved (71.42%) in a study by Misra et al[35]on 14 cases of JE.

In this study Thalamus involvement was noted in both the cases of Japanese encephalitis (Fig 9) in concordance with the study of Misra et al[35].

In common with previous reports (Snyder et al[36], Cabral et al[37]), clinically insignificant ventriculomegaly and/or subarachnoid space widening were the most common abnormalities seen in this study in cases of pyogenic meningitis. Subdural effusions were identified in one patient. Subdural effusion can be demonstrated in upto 45 % of children with bacterial meningitis according to Snedeker et al[38]. A brain abscess following meningitis is extremely rare according to studies by Cabral et al[37]and Kline et al[39]. According to Packer et al[40], intracranial empyemas are seen in less than 2 percent of patients and tend to occur late in the course of the illness or after treatment has been stopped. According to Suchet et al[41], CT has not been found to be entirely reliable in distinguishing such empyemas from benign subdural effusion and clinical setting is important in assessing such a case.

CT failed to demonstrate 1 cerebral edema, 1 subtle cerebral infarct and 1 subdural effusion finding. Overall CT failed to demonstrate 1 case of pyogenic meningitis (Fig 10) that is detected by MRI.

According to a study by Hyderman RS et al[42] on 30 children with bacterial meningitis, CT is insensitive in identifying the subtle changes that occur in bacterial meningitis. MRI reveals lesions in CT negative cases and more extensive disease in CT positive cases.

Conclusion

This study was performed to determine the burden of infective causes of epilepsy which can be detected on CT and MRI. Radiological imaging in the form of CT and MRI play a vital role in etiological evaluation in cases of epilepsy. No doubt that MRI is the first choice in case of epilepsy. MRI is also recommended, as it is a radiation free modality.

Infections constitute one of the important cause of epilepsy. For characterization of granulomas, MRI is better than CT, and CT is superior in identifying calcified lesions.

Meningitis is an important cause among infections other than inflammatory granuloma.

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