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Radiology

ROLE OF IMAGING IN STATUS EPILEPTICUS- A PROSPECTIVE OBSERVATIONAL STUDY IN A TERTIARY HOSPITAL

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ABSTRACT Status epilepticus is a condition that can have long term consequences including neuronal death, neuronal injury and alteration of neuronal networks depending on the type and duration of seizures. It can occur in all age groups. Hence it is important to understand the underlying cause of status epilepticus which helps in appropriate treatment. Our prospective observational study emphasizes on imaging, causative actors associated with convulsive status epilepticus and usefulness of doing MRI over CT in emergency situations like status epilepticus.

KEYWORDS : Status epilepticus, Encephalitis, Granuloma, Glioma.

INTRODUCTION

Status epilepticus is defined as continuous seizures or repetitive, discrete seizures with impaired consciousness in the interictal period. The duration of seizure activity to meet the definition has traditionally been 15–30 min [1]. Spontaneous remission of seizure is unlikely if it persisted for 5min; therefore for operational definition of SE more than 5 min duration has been suggested [2]. Epilepsy is a chronic condition characterized by recurrent seizures unprovoked by an acute systemic or neurological insult [3] According to international league against epilepsy 2015, the proposed new definition of SE is as follows: Status epilepticus is a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms, which lead to abnormally, prolonged seizures (after time point t1). It is a condition, which can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures. This definition is conceptual, with two operational dimensions: the first is the length of the seizure and the time point (t1) beyond which the seizure should be regarded as "continuous seizure activity." The second time point (t2) is the time of ongoing seizure activity after which there is a risk of long-term consequences. In the case of convulsive (tonic-clonic) SE, both time points (t1 at 5 min and t2 at 30 min) are based on animal experiments and clinical research.

For the purpose of study >5 minutes of continuous seizure or 2 or more discrete seizures between which there is incomplete recovery of consciousness is considered as status epilepticus.

Status epilepticus (SE) might occur: a) as a consequence of chronic epilepsy or febrile convulsions in one third of cases, b) as new onset epilepsy in one third and c) as a complication of acute encephalopathy in one third[8]. The underlying etiology, pathophysiology and treatment vary with the type of patients.

MRI is a useful imaging technique to find out the underlying cause of CSE, especially potentially treatable ones like infectious, inflammatory, vascular and neoplastic etiologies, thereby helping in taking appropriate treatment decisions [9]. Status epilepticus per se can cause brain damage with imaging abnormalities and is also useful in predicting the outcome.

CT brain is generally obtained in patients with status epilepticus for any pathology. However the role of MRI in evaluation of patients with status epilepticus is still not unclear. Also there is no consensus as to whether MRI should be carried out in all the patients with status epilepticus or whether it should be limited to a specific subgroup of patients.

The present study emphasizes on identifying various abnormalities on imaging, causative factors associated with convulsive status epilepticus and usefulness of doing MRI over CT in emergency situations like status epilepticus.

Aims And Objectives Of The Study

1. To detect various abnormalities on MRI and study the etiological factors in patients presenting with convulsive status epilepticus.
2. To assess the diagnostic value of MRI over and above that provided by CT in emergency situations like convulsive status epilepticus.

Classification

A new diagnostic classification system of SE is proposed by International league against epilepsy, which will provide a framework for clinical diagnosis, investigation, and therapeutic approaches for each patient. There are four axes: semiology; etiology; electroencephalography (EEG) correlates; and age.[24]

Based On Semiology

(A) With prominent motor symptoms

Convulsive SE (CSE, synonym: tonic–clonic SE)

1. a. Generalized convulsive
1. b. Focal onset evolving into bilateral convulsive SE

A. 2 Myoclonic SE (prominent epileptic myoclonic jerks)
Focal motor
Tonic status
Hyperkinetic SE

(B) Without prominent motor symptoms (i.e., nonconvulsive SE, NCSE)
NCSE with coma (including so-called "subtle" SE)
NCSE without coma
Generalized
Focal

Based on Etiology of status epilepticus

1. Known (i.e., symptomatic)
2. Acute (e.g., stroke, intoxication, malaria, encephalitis, etc.)
3. Remote (e.g., posttraumatic, postencephalitic, poststroke, etc.)
4. Progressive (e.g., brain tumor, Lafora's disease and other PMEs, dementias)
5. SE in defined electroclinical syndromes

Neurocysticercosis

A cysticercus cyst in brain parenchyma typically goes through four stages of involution.

The vesicular stage :

In this stage, the cyst has a thin wall and the cyst fluid has signal intensity similar to that of CSF in all sequences. In viable cysts, an eccentrically located mural nodule represents the scolex. No contrast enhancement or edema seen.

FLAIR images detect a significantly higher number of scolices than

other sequence which is helpful for diagnosis of NCC. FLAIR image show the scolex as an eccentrically punctuate bright signal intensity within the cysts.

In the **colloidal stage**, cyst is slightly hyperdense to CSF on CT and ring enhancing capsule on CECT. On MRI cystic fluid has a signal intensity slightly distinct from that of CSF in all sequences. The scolex begins to show signs of degeneration, and a gradual decrease in the size of the lesion is noticed. Contrast enhancement usually peripheral, and perilesional edema are seen.

Granular nodular stage, the parasite is already dead and retracted, the lesion begins to mineralize. There is nodular or ring enhancement of the lesion, and edema is less extensive.

In the **nodular calcified stage**, the lesion is completely mineralized. These lesions appear as small nodules with hypointensity on T2-weighted and T2* – weighted images, better detected on CT studies. Some calcified lesions present persistent contrast enhancement on MR imaging, others may lead to active inflammation during therapy.[17]

Tuberculoma

CT shows one or more iso to slightly hyperdense round lobulated lesions with variable perilesional edema. The MR features of the individual tuberculoma depend on whether the granuloma is non-caseating or caseating with a solid center, or caseating with a liquid center. The non-caseating granuloma is usually iso/hypointense on T1W and hyperintense on T2W images. These granulomas show homogeneous enhancement after injection of contrast agent. The caseating solid granulomas appear relatively isointense/hypointense on T1W images with isointense/hyperintense rim and isointense to hypointense on T2W images [20]. These lesions show rim enhancement on post- contrast T1W imaging. The granulomas with central liquefaction of caseous material appear centrally hypointense on T1W and hyperintense on T2W images and show rim enhancement after contrast administration. MR Spectroscopy has been found to be specific for intracranial tuberculomas when combined with imaging. Intracranial tuberculomas are characterized by a spectral pattern that primarily involves long chain lipids, with a 0.9 to 1.6 ppm peak range, associated with a virtual absence of all brain metabolites normally present.

Meningitis

In early meningitis, the CT or MR findings may be normal. On contrast enhanced CT scan, there is enhancement of the inflammatory exudate in the involved basal cisterns, fissures or sulci . The abnormal leptomeningeal contrast enhancement is typically more readily apparent and more intense on MR imaging rather than on CT scanning. MR imaging also easily differentiates basal cisternal enhancement from vessels in the region of the circle of Willis in a patient with CT findings of equivocal abnormal subarachnoid space enhancement. In addition, contrast enhancement over the cerebral convexities is easier to appreciate on MR imaging as opposed to CT scanning because the overlying inner table of the skull is seen as an adjacent signal void on MR imaging. In tuberculous meningitis tuberculomas along with the exudates are found in the meninges which are composed of cellular infiltrate degenerated and partly caseated fibrin, tubercles and rarely bacilli accounting for the visibility on the precontrast MT T1-weighted. Dural (i.e. pachymeningeal) enhancement follows the inner contour of the calvaria, whereas pial- subarachnoid (i.e. leptomeningeal) enhancement extends into the depths of the cerebral and cerebellar sulci and fissures.

Encephalitis

Herpes simplex-

CT is often normal early in the course of disease. Hypodensity with mass effect in one or both temporal lobes may be seen.T1 scans show gyral swelling with indistinct graywhite interfaces. T2 scans demonstrate cortical/subcortical hyperintensity with relative sparing of the underlying white matter. FLAIR is the most sensitive sequence and may be positive before signal changes are apparent on either T1- or T2WI. Bilateral but asymmetric involvement of the temporal lobes and insula is characteristic of HSE but is not always present. T2* (GRE, SWI) may demonstrate petechial hemorrhages after 24-48 hours. Gyriform T1 shortening, volume loss, and confluent curvilinear "blooming" foci on T2* are seen in the subacute and chronic phases of HSE.

Japanese encephalitis-

MRI shows areas of hyperintensity on T2-weighted images involving the thalamus and basal ganglia. Involvement of thalami and basal ganglia is usually seen with or without involvement of other regions.

ADEM-

Lesions associated with ADEM are typically bilateral but may be asymmetric and tend to be poorly marginated. Almost all patients have multiple lesions in the deep and subcortical white matter, while the periventricular white matter is generally spared. The thalami and basal ganglia are frequently affected, and lesions in these locations are often symmetrical. Brainstem and spinal cord abnormalities on MRI are common in ADEM. The number of lesions varies, and their diameters range from <5 mm to 5 cm. In the spinal cord, large confluent intramedullary lesions that extend over multiple segments are common, and the degree of contrast enhancement is variable

Vascular Lesions: Cerebral Infarction

Intracerebral haemorrhage associated with highest incidence of post stroke seizures and transient ischemic attack is associated with lowest incidence (3%).[28,29] Seizures after haemorrhagic strokes due to irritation caused by products of blood metabolism. Late onset seizures are associated with persistent changes in neuronal excitability and gliotic scarring.

Gliosis

Gliosis is an astrocytic response to tissue damage and is the end result of various focal or diffuse central nervous system injuries trauma, infection, infarctions which can be focal / diffuse.[19]Gliosis appears bright on T2 as well as FLAIR, unlike encephalomalacia which follows CSF signal on all sequences.

Mesial Temporal Sclerosis And Temporal Lobe Epilepsy[37,38]

The long-standing hypothesis proposing a causal link between TLE, MTS and Prolonged febrile seizures (PFS) was first articulated by Sommer in 1880. MTS is the most common lesion found in TLE and, unless there is dual pathology, removal of the affected hippocampus and temporal lobe has a high chance of eliminating the seizures. Furthermore MTS is rarely found in people without seizures, suggesting that the presence of MTS can be used as a marker for TLE, even in the absence of reported clinical seizures.

Imaging features of MTS:

Primary signs :

- A small atrophic unilateral hippocampus
- Hyperintensity on both T2W and FLAIR images.
- Loss of hippocampal internal architecture and that of normal digitations of the head.

Secondary signs:

- Unilateral atrophy of the mamillary body, fornix columns (circuit of papez) and the amygdala.
- Increased T2W signal in the anterior temporal lobe white matter with loss of grey-white demarcation in the ipsilateral anterior temporal lobe.
- Unilateral dilatation of the temporal horn (a less reliable secondary sign).
- Unilateral atrophy of the collateral white matter bundle.[75]

Volumetric MR imaging:

Quantitative evaluation of hippocampal volume has been found to marginally increase the sensitivity over visual analysis in detection of hippocampal sclerosis.

Measuring size can be accomplished by manually tracing the hippocampus. The normal, ipsilateral hippocampal volume is approximately 2.8 MI.

Postictal Imaging

Prolonged ictal activity induces hypermetabolism with increased glucose utilization. Perfusion increases but is still insufficient to match glucose demand. The result is compromised cellular energy production, cytotoxic cell swelling, and vasogenic edema. With prolonged severe seizure activity, the blood-brain barrier may become permeable, permitting leakage of fluid and macromolecules into the extracellular spaces.

General Imaging Features[32]:

Imaging findings in SE vary with acuity and severity. Most acute periictal abnormalities are reversible and normalize within a few days.

Irreversible changes do occur, especially with generalized convulsive SE

Ct Findings:
Initial NECT scans may be normal or show gyral swelling with sulcal effacement and parenchymal hypodensity. CECT may demonstrate gyral enhancement in a nonvascular distribution.

MR FINDINGS: Periictal MR shows T2/FLAIR hyperintensity with gyral swelling. The subcortical and deep WM is relatively spared. Crossed cerebellar diaschisis, ipsilateral thalamic involvement, and basal ganglia lesions are seen in some cases. Gyriform enhancement on T1 C+ varies from none to striking. Diffusion restriction with uni- or bilateral hippocampal, thalamic, and cortical lesions is common.

Scans performed a week to several months following SE disclose structural abnormalities in approximately one third of patients. Reported permanent abnormalities include focal brain atrophy, cortical laminar necrosis, mesial temporal sclerosis, and lower fraction anisotropy in normal-appearing WM.

MATERIALS AND METHODS
This is a prospective observational study performed over 97 patients who had convulsive status epilepticus and admitted under departments of General Medicine, paediatrics and neurology.

The study extended over a period of twelve months from April 2021 to April 2022. The cases were evaluated in the department of Radiodiagnosis. All the patients who already had their CT done were taken up for MRI & was performed on SIEMENS MAGNETOM AVANTO 18 CHANNEL 1.5 T MRI.

All cases were done within 24hrs of after seizure onset. Diagnosis drawn based on MR findings is noted and its diagnostic value is assessed over and above that provided by CT.

Patient selection:
97 patients (from age group 1yr-80yrs) were included in our study who presented with convulsive status epilepticus. Patients who had CSE during the period of hospital stay were also included in our study. Clinical, laboratory and CT imaging findings of the Subjects were recorded and then taken up for MRI. Informed consent was taken from all patients.

Exclusion criteria:
- Acute head injury patients
- Pregnancy
- Patients who are on ventilator support
- Critically ill patients where MRI cannot be done due to patient condition/ethical issues.

Patients with pre-existing renal failure, claustrophobia and with other contra indications for MRI were excluded from the study.

Contra indications for MRI:
1. Cardiac pacemaker or artificial heart valve
2. Metal plate, pin, or other metallic implant
3. Piercings (particularly body piercing)
4. Intrauterine device, such as Copper-7 IUD
5. Insulin or other drug pump
6. Aneurysm clips
7. Previous gunshot wound
8. Cochlear implant or other hearing device
9. Employment history as a metalworker (had metal in eye)
10. Permanent (tattoo) eye-liner

Technique :
All patients, screened before entry into the MRI scanning room for ferromagnetic objects, cardiac pacemakers, aneurysm clips etc.

Patients were examined in the supine position on the MRI machine , proper positioning, and immobilization of the head was obtained.

MRI was performed using 4 channel HEAD COIL. A single-slice, three-axis localizer scan was done followed by 5mm slice thickness of Axial T1 (TR 468, TE 11), Axial T2 (TR 4500, TE 111), sagittal T2 (TR 4000 TE 110), Coronal T2 (TR 4500, TE 124), Axial FLAIR (TR 9500 TE 102), Axial DWI (TR 3500, TE117), Axial GRE (TR 779, TE 25)

Epilepsy protocol is done in cases where there is no detectable acute symptomatic cause for seizures. Sequences used are:
- T1 INVERSION RECOVERY (IR)CORONAL OBLIQUE-2MM SLICES (TR 5680, TE 68, TI 760)
- T2 CORONAL OBLIQUE-2MM SLICES (TR 4260, TE 114)
- T2 INVERSION FLAIR CORONAL OBLIQUE- 2MM SLICES (TR 5000, TE 106, TI 2300)

If a focal lesion or tumour is present, Gadoversetamide (Multihance) was injected intravenously and FAT SAT Coronal T1 (TR 300, TE 70), FAT SAT Sag T1 (TR 500, TE 15) and FAT SAT axial T1 (TR 755, TE 111) were obtained.

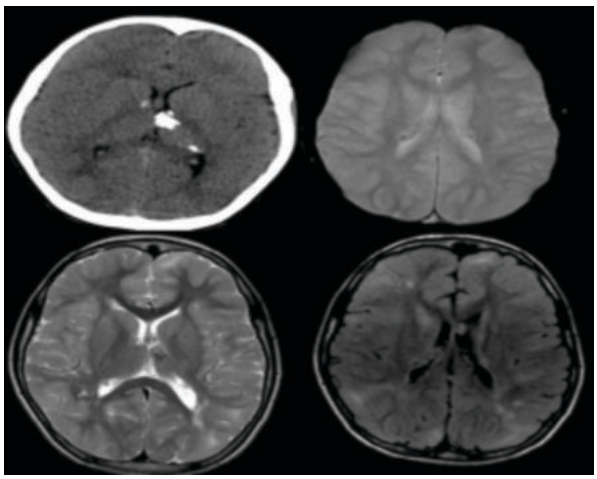


Figure 1. Axial CT shows periven ricular calcifications. MRI showing T2/FLAIR subcortical hyperintensities in frontal and parietal region with few subcortical tubers-Tuberous sclerosis in a 11 year male child.

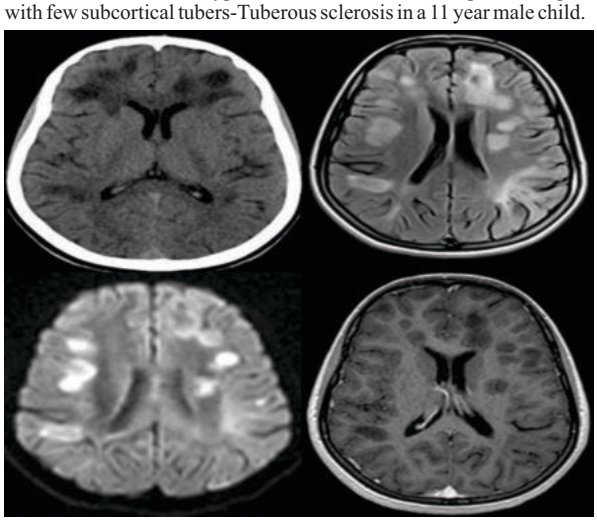


Figure 2. *CT showing hypodensities in bifrontal and right parietal white matter.MRI showing multiple fluffy T2/FLAIR hyperintense lesions in frontoparietal lobes in subcortical location showing diffusion restriction with no enhancement on contrast-ADEM*

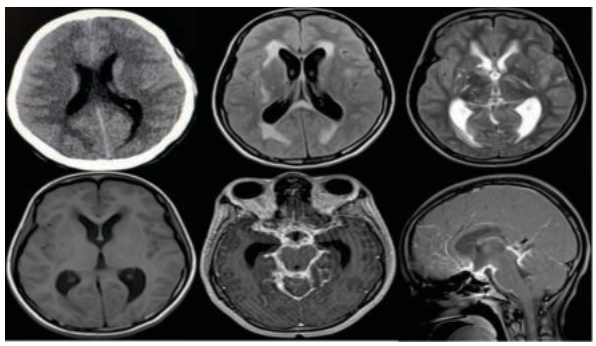


Figure 3. *CT showing mild prominence of ventricles.Axial T2/FLAIR showing dilated ventricles with periventricular seepage of CSF and enhancing basal cisterns-meningitis.*

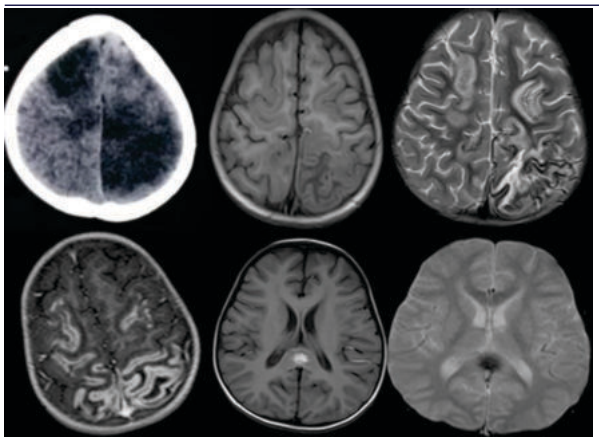


Figure 4.CT showing bilateral frontoparietal hypodensities. MRI T1 hypointensity,T2 hyperintensity in bilateral high frontoparietal lobes showing gyral enhancement.T1hyperintensity in splenium of corpus callosaum showing booming on GRE-Hemorrhagic Encephalitis.

Table1. Age incidence

Age in years	Number of cases	Percentage
1-15	28	28.8
16-30	13	13.4
31-45	18	18.5
46-60	20	20.6
>60	18	18.5
Total	97	100

In our study, Status epilepticus is more common in less than 15yrs age group and more than 45 years age group

Table2. Sex incidence

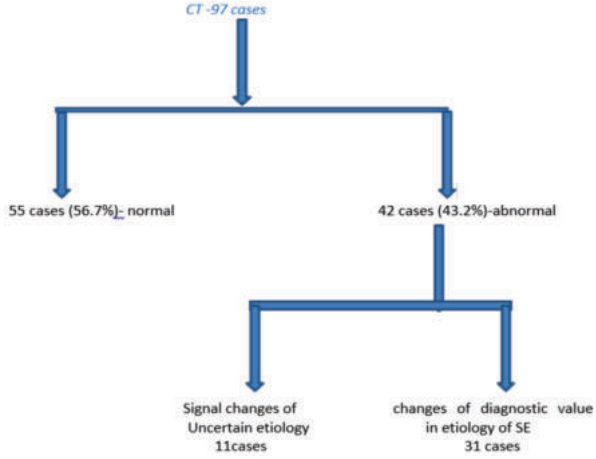
Sex	No. of cases	Percentage distribution
Male	56	57.7
Female	41	42.2
Total	97	100

In our study majority were male patients accounting for about 58%

Table3. MRI abnormalities in patients with partial and generalized SE

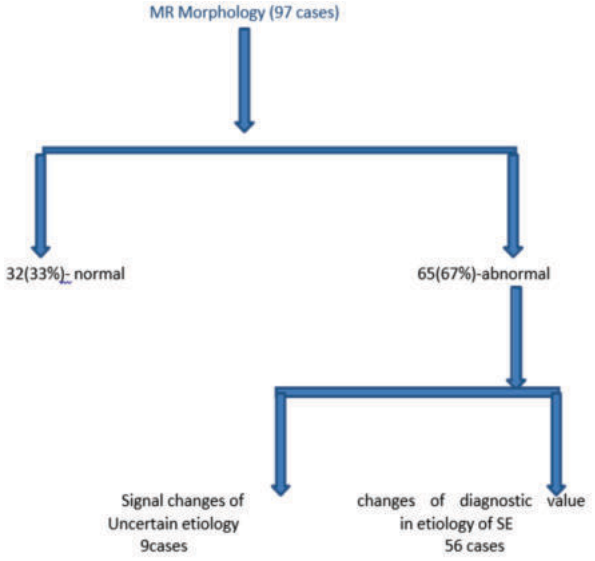
MRI features	Partial SE	Generalized SE
MRI with findings	30	35
Normal MRI	9	23
Total	39	58

Among 39 patients with partial SE, 30 had lesions detected on MR imaging with a diagnostic yield of 70%. In generalized SE group, the diagnostic yield is 50%.



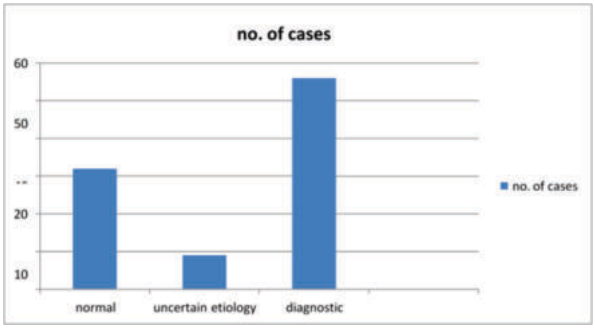
Flowchart showing CT imaging and abnormalities in SE

Of the 97 cases on CT, 55 were normal and 42 were abnormal but the etiology could be known only in 31 cases (32%).



Flowchart representing imaging abnormalities on MRI

Of the 97 cases on MRI 32 cases are normal and 65 cases are abnormal. Of the 67 abnormal cases, 9 cases showed signal changes of uncertain etiology or uncertain significance and the etiology could be known in 56 cases.



Bar diagram depicting MR morphology in our study

Table 4: Comparative evaluation of CT and MRI in diagnosing etiology of SE.

Status epilepticus	MRI	CT
Etiology known	56	31
Etiology unknown	41	66
Total	97	97

At 95% confidence interval, chi square value is 13.02, with **p-value <0.01** which is highly significant. MRI has better diagnostic value in terms of etiology when compared to CT, as p-value is highly significant.

Table5: Abnormalities on CT and MRI in present study

	MRI	CT
Abnormal	65	42
Normal	32	55
Total	97	97

At chi square =11.02, **p-value is <0.01** which is highly .

Table 6. Overall distribution of patients based on MRI abnormalities

MRI abnormality	No. of cases
Neuro infections	14
Gliosis	14
Non specific signal changes	9
Vascular causes	8
Neoplastic	4
MTS	3
ADEM	2
PRES	2

NKHC	1
Cavernoma	1
Cerebral edema	1
Tuberous sclerosis	1
Adrenoleukodystrophy	1
Heterotopia	1
Cortical laminar necrosis	1
Precentral gyral hyperintensity	1
Seizure induced gyral hyperintensity	1
Total	65

Neuroinfections are most common acute symptomatic etiology accounting for about 14.5 % of cases Among remote symptomatic etiology, gliosis is the most common cause

DISCUSSION

Status epilepticus (SE) is an acute neurological emergency with significant mortality and morbidity, if not promptly and vigorously treated. Data on the yield of neuroimaging in patients with status epilepticus is limited. Emergent neuroimaging may require moving an ill patient, limit access to the patient if they were to deteriorate, possibly expose that patient to the risks of ionizing radiation[66] and/or sedation and delay other management. Conversely, the clinical assessment of these patients is challenging as they are post- ictal, sedated from anti-epileptic medications and frequently intubated. Therefore, a significant enough proportion of these patients should have clinically urgent or emergent findings to justify performing emergent imaging on a routine basis. CT brain is generally obtained in patients with status epilepticus for any pathology. However the role of MRI in evaluation of patients with status epilepticus is still not unclear. MRI is a useful imaging technique to find out the underlying cause of SE, especially potentially treatable ones like infectious, inflammatory, vascular and neoplastic etiologies, thereby helping in taking appropriate treatment decisions[9].

Our study includes 97 cases of status epilepticus and age group ranged from 16mnths to 72yrs.28 cases are from pediatric age group below the age of 15yrs.

In present study focal seizures were seen in 39 patients, of which etiology could be established in 27 patients i.e. the diagnostic yield is 70%.

Table 1. Focal SE in various studies

Study	No. of patients	No. of positive cases on MRI
Sinha et al.	11	11
M.K.Goyal et al.10	7	4
Present study	39	27

MRI and the etiology of status epilepticus

Neuroinfections is the most common abnormality detected on MRI in developing countries. While in the western studies [8], neuroinfections account for only 9% of total cases of status epilepticus and stroke is the leading cause among the acute symptomatic cases, accounting for 14–22% of SE in adults (DeLorenzo et al., 1995; Knake et al., 2001). In older adults, remote stroke is a major cause.

In present study neuroinfections are most common (14.4%) followed by vascular (8.2%), neoplastic (4.1%), ADEM (2%) & PRES (2%) Among 14 cases of neuroinfections granulomas are seen in 7 patients of which tuberculomas are seen in 4 cases and are diagnosed on the basis of hypo-isointense lesion on T1WI and hypointense to variable intensity lesion with surrounding edema on T2WI, showing ring enhancement on post contrast images. Routine chest radiograph was also performed for these patients, 3 of them showing positive signs of tuberculosis.

Four cases of meningitis in our study were diagnosed on the basis of leptomeningeal enhancement with hydrocephalus. CSF analysis done in 2 patients showed increased protein and neutrophils with decreased glucose in one patient and normal in another patient.

Two cases of encephalitis in our study, of which one was positive for dengue serology where there was bifrontal T2/FLAIR hyperintensity with restricted diffusion and gyriform pattern of enhancement post contrast.. The other case was of herpes simplex -1 with typical MR imaging features of T2 hyperintensity of affected white matter and cortex, showing variable enhancement and restricted diffusion(due to cytotoxic edema)which was confirmed by PCR(polymerase chain reaction)DNA test which reveals even tiny amounts of viral DNA ,being the diagnostic test for encephalitis Vascular causes are most common abnormality in western countries. Infarcts contribute to 8 cases in our study of which 5 were venous and 2 are arterial. Diagnosis of stroke should be considered in patients presenting with SE and focal neurological deficit. Emergent neuroimaging should be considered in patients with postictal neurological deficit. Intra cranial haemorrhage is associated with high incidence of post stroke seizures and transient ischemic attack is associated with lowest incidence.

Tumours were identified as a cause of SE in 4 (7%) cases. All of them are supratentorial in location , 2 of them were gliomas in frontal lobe and left parietal lobe, one case each of oligodendroglioma and ganglioglioma in temporal lobe. Subsequent biopsy and surgery was done which confirmed the diagnosis. Most of the tumours that present with seizures are frontal in location. MRI has nearly 100% sensitivity for the identification of neoplastic lesions.

The most common lesion identified in remote symptomatic group is gliosis(14.4%). Gliosis is an astrocytic response to tissue damage and is the end result of various focal or diffuse central nervous system injuries trauma, infection, infarctions which can be focal / diffuse. In the present study of 14 cases, gliosis is the result of hypoxic ischemic insult in 4, infarct in 5, trauma in 3, postsurgery in 2.

3 cases of MTS were identified in our study and were diagnosed based on the principal findings of volume loss and abnormal signal in the hippocampus with identification of loss of hippocampal architecture on inversion recovery sequence. In the present study, hippocampal sclerosis was identified in 3% patients and all of them were known case of epilepsy. Similar results were shown by Lefkopoulos et al in their study of 120 patients with seizures. Minor findings such as atrophy of the ipsilateral fornix and mammilary body, ipsilateral dilatation of the temporal horn were also identified in our patients.

We report a case of phakomatosis that was tuberous sclerosis in a 11year old male, who presented with seizures and skin manifestations. MRI, showed subependymal calcified nodules, cortical tubers and non-specific white matter changes.

Overall in 56 cases of etiology known on MRI, 35 cases (63%) are of acute symptomatic etiology, 21 cases belong to remote symptomatic etiology (37%). Thus MRI contributes significantly in evaluating the cause of SE and probably treatment could be initiated or modified in 35 cases of acute symptomatic etiology.

CT and the etiology of Status epilepticus

CT is generally done in patients presenting with SE to rule out any emergent intracranial pathology and can detect abnormalities in which earlier treatment may improve outcomes. Of the 97 cases on CT, 55 were normal, 11 cases showed insignificant findings or findings of uncertain diagnosis and the exact etiology could be known only in 31 cases.

Among the 31 cases, 17 patients had acute symptomatic cause which includes suspected meningitis with hydrocephalus in 2, granulomatous lesions in 4, infarct in 6, tumours in 2 & one case each of cerebral edema, PRES & ADEM. In remote symptomatic causes gliosis is the most common etiological substrate found which is seen in 12 cases. A case of heterotopia in left medial temporal lobe has also been reported in our study. CT has no much role in diagnosis of other remote symptomatic causes.

Several studies had addressed the utility of neuroimaging in status epilepticus with abnormal CT imaging findings varying from as less as 18%(Jan M et al.[74]) to 48% (Pradeep P Nair et al.). The present study reports abnormal CT neuroimaging in 43% of cases. CT is easily available, requires less imaging time & can detect abnormalities which improves patient outcome in emergency situation. In present study CT has shown to be useful in emergency situation in 17.5 % cases.

Efficacy of MRI over CT in the evaluation of etiology of SE

The choice of imaging modality is often debated, depends on urgency, availability, and resolution. However, CT confers radiation exposure that may not be trivial especially for the youngest children. However, several factors such as time constraints, cost factor, availability and long duration required to carry out the MRI might limit its utility and widespread application.

In present study CT could contribute to diagnosis of etiology of SE in 31 patients whereas MRI could contribute to diagnosis in 56 cases**.** Overall MRI improved the diagnosis in 25 of 97 patients (25.7%) over and above that provided by CT, clinical information and laboratory values. CT was helpful in identifying acute vascular lesions and acute edema, whereas MRI was superior in identifying subtle abnormalities and remote symptomatic etiologies such as dysplasia and mesial temporal sclerosis.

In the present study MRI improved the diagnosis in 12 cases of acute symptomatic etiologies, of which 2 cases of meningitis which on plain and contrast CT were normal but MRI showed leptomeningeal enhancement. The abnormal leptomeningeal enhancement is typically more apparent and intense on MR imaging rather than on CT imaging. In addition, contrast enhancement over the cerebral convexities is easier to appreciate on MR imaging as opposed to CT scanning because the overlying inner table of the skull is seen as an adjacent signal void on MR imaging. Overall in 4 cases of meningitis CT was normal in 2 cases and the others showed hydrocephalus.

In 2 cases with GCSE, hypodensity on CT was interpreted as postictal edema. However MRI revealed ring lesions suggestive of granuloma. Another case of granuloma which is seen on MRI was not picked on CT. FLAIR images and ciss 3d sequences are better in demonstrating scolex on MRI. In 7 cases of granulomatous lesions picked up on MRI, CT is suggestive of the same diagnosis in only 4 patients.

One suspected case of encephalitis which was normal on CT has shown focal cortical and subcortical signal intensity changes in left temporal lobe attributable to focal cerebritis. Another case of bifrontal hypodensities of unclear etiology was diagnosed as encephalitis on MRI which showed restricted diffusion with T2/FLAIR hyperintensity in bilateral frontoparietal lobes consistent with encephalitis.

Overall MRI is useful in emergency situation and treatment could be modified in 20% of cases in our study. Thus, carrying out MRI in emergency situation in the setting of SE was useful in diagnosis as well as treatment. However, several factors might limit its utility and widespread application. We recognize that patients with SE are frequently unstable and MRI may need to be deferred until clinical status has improved. Because CT scan can detect abnormalities in which earlier treatment may improve outcomes, CT can be useful in unstable patients in whom MRI cannot safely be performed in a reasonable time interval.

In remote symptomatic etiologies 14 cases of gliosis are seen of which 12 were positive on CT indicating that CT is as sensitive as MRI in detecting gliotic changes. In other remote symptomatic etiologies, MRI is far superior to CT. The present study reports 3 cases of MTS, a case of heterotopia & adrenoleukodystrophy. Normal conventional MRI sequences has low sensitivity and specificity for the diagnosis of MTS. A study done by Sancrez-Alvarez JC et al. in 2000 concluded that a combination of inversion recovery T1W images & T2 relaxometry optimize MTS diagnosis in most cases.

Table 2. Various studies on percentage abnormalities picked on CT and MRI in status epilepticus.

Study	No. of cases	Abnormal CT(%)	Abnormal MRI(%)
R.K.Singh et al.11 (new onset SE)	44	20	56
Pradeep P Nair et al.	44	47.7	63
Jan M et al.74 (CSE in intractable epilepsy)	18	18	55
M.K.Goyal et al.10	34	38	70
Tracey A Milligan et al.	76	-	84
Present study	97	43	67

In our study percentage abnormalities on MRI correlated well with the previous studies. A relative higher incidence of abnormalities on CT may be due to selection bias, referral bias where there are more number of missed cases of drug defaulters who present with SE and do not undergo neuroimaging.

We also calculated the p value comparing the CT and MRI in terms of etiology known or unknown**. At 95% confidence interval, chi square value 13.02 and p-value is <0.001 which is highly significant**.

Thus MR imaging detected abnormalities and determined etiology in a significant proportion of patients with status epilepticus over and above that provided by CT and so MRI is very useful in diagnosis and management even in emergency situations like status epilepticus.

Status epilepticus in epilepsy

In the context of epilepsy, SE may develop in those with a previous diagnosis of epilepsy or de novo, as its initial manifestation. Approximately 15% of patients with epilepsy have had at least one episode of status during their lifetime. Most often, the SE is due to the epilepsy itself, triggered by medication nonadherence, resulting in subtherapeutic AED levels (Aminoff & Simon, 1980) or by inappropriate drug treatment(Thomas et al., 2006). Approximately 12%of patients who eventually develop epilepsy have presented with SE as their first clinical manifestation (Hauser, 1990)[9]. In these patients, SE may be an intrinsic manifestation of disease, sometimes with recurrent episodes, or the epilepsy may be the consequence of a prolonged SE, with neuronal death and alteration of networks causing recurrent seizures after the initial event.

In present study MRI did not contribute to etiology in vast majority of patients with past history of epilepsy, suggesting that MRI yield may be very low in this subgroup of patients.

CONCLUSION

To conclude, MR imaging detected abnormalities and determined etiology in significant proportion of patients with CSE. However patients with SE are frequently unstable and MRI may need to be deferred until clinical status is improved. CT can be useful in patients in whom MRI cannot be performed in reasonable time interval. When available and safe, MRI should be the imaging modality of choice even in emergency setting.

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