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Original Research Article

A Study on Between Early Versus Delayed Neuroimaging in Trauma Acute Encephalopathy in Paediatric Age Group

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Abstract:

Introduction: Encephalopathy can present a very broad spectrum of symptoms that range from mild, such as some memory loss or subtle personality changes, to severe, such as dementia, seizures, coma or death. In general, encephalopathy is manifested by an altered mental state that is sometimes accompanied by physical manifestations.

Materials & Methods: MRI/CT brain will be performed according to the clinical requirement and patient condition at initial admission. If there is clear diagnostic information on initial imaging no further neuroimaging will be performed on these children unless a prognostic imaging is deemed necessary by treating neurologist. Delayed imaging will be done when the initial neuroimaging is normal/nondiagnostic and child continues to have neurological symptoms and signs without obvious diagnoses from clinical, blood, CSF investigations.

Results: Highest percentage of symptom was fever (68.29%) followed by seizure (65.85%) and encephalopathy (60.9%). Vomiting was in 34.14% of patients. Lowest percentage of symptom was Visual impairment. (21.95%). In 39.02 % of cases, first neuroimaging was done on Day-2 of illness and in 26.82% of cases, it was done on Day-1 of illness followed by 12.19% of patients on 3rd day of illness.

Conclusion: Consequently, paediatric neurologists may utilize several different tests at the same time to diagnose both the primary condition (the cause of encephalopathy) and the encephalopathy itself.

Keywords: Encephalopathy, Illness, Paediatric.

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Introduction

Encephalopathy can present a very broad spectrum of symptoms that range from mild, such as some memory loss or subtle personality changes, to severe, such as dementia, seizures, coma or death. In general, encephalopathy is manifested by an altered mental state that is sometimes accompanied by physical manifestations. Regardless of aetiology most patients with CNS infection have similar clinical manifestations. Common symptoms include fever, headache, nausea, vomiting, anorexia, restlessness, altered state of consciousness and irritability, whereas common signs include photophobia, neck rigidity, obtundation, stupor, coma, seizures & focal neurologic deficits. The severity & constellation of signs are determined by the specific pathogens, the host & the area of the CNS affected. Infection of the CNS may be diffusing or focal. Meningitis and encephalitis are examples of diffuse infections. Meningitis implies primary involvement of the meninges, whereas encephalitis indicates brain parenchymal involvement. Because these anatomic boundaries are often not distinct, many patients have evidence of both meningeal & parenchymal involvement and should be considered to have meningoencephalitis. Bacterial meningitis is one of the most potentially serious infection occurring in infants & older children. This infection is associated with a high rate of acute complications & risk of long term morbidity. The incidence of bacterial meningitis is so high in children that it should be included in the differential diagnosis of all children with altered mental status and other evidence of neurologic dysfunction. The causes of bacterial meningitis in neonatal period include group B and D streptococci, gram negative enteric bacilli and Listeria monocytogenes. The most common causative organism of bacterial meningitis in children between 2 months to 12 years include streptococcus pneumonia & H. influenza type b.62.Risk of Tubercular meningitis is invariably present in children. Risk of developing Tubercular- meningitis following miliary tuberculosis is maximum in first 3 months of acquiring primary complex due to haematogenous spread of Tuberculous organisms.6 A major risk factor for meningitis is the lack of immunity to specific pathogenic organisms associated with young age. Additional

risk factors include recent colonization with pathogenic bacteria, close contacts with individuals having infective disease e.g. Tuberculosis, overcrowding, poverty. The mode of transmission is probably person to person contact through respiratory tract secretions or droplets. Specific host defence defects due to altered immunoglobulin production in response to encapsulated pathogens may be responsible for increased risk of bacterial meningitis. Congenital or acquired CSF leak across a mucocutaneous barrier such as cranial or midline facial defects, chronic supportive otitis media or CSF leakage through a rupture of the meninges due to basal skull fracture is associated with an increased risk of pneumococcal meningitis.

Materials & Methods

Study Population: Children between 1-month and18 years of age.

Inclusion Criteria

Patients meeting the following criteria will be included into the study:

1. Children between 1-month and18 years of age.
2. Children with status epilepticus and acute encephalopathy.
3. Children with acute encephalopathy with GCS ≤13

Exclusion Criteria

Patients meeting ANY of the following exclusion criteria will be excluded from the study:

1. Children below 1 month and above 18 years of age.
2. Children with history of head trauma.
3. Children with previous non-progressive structural anomalies
4. Toxin ingestion

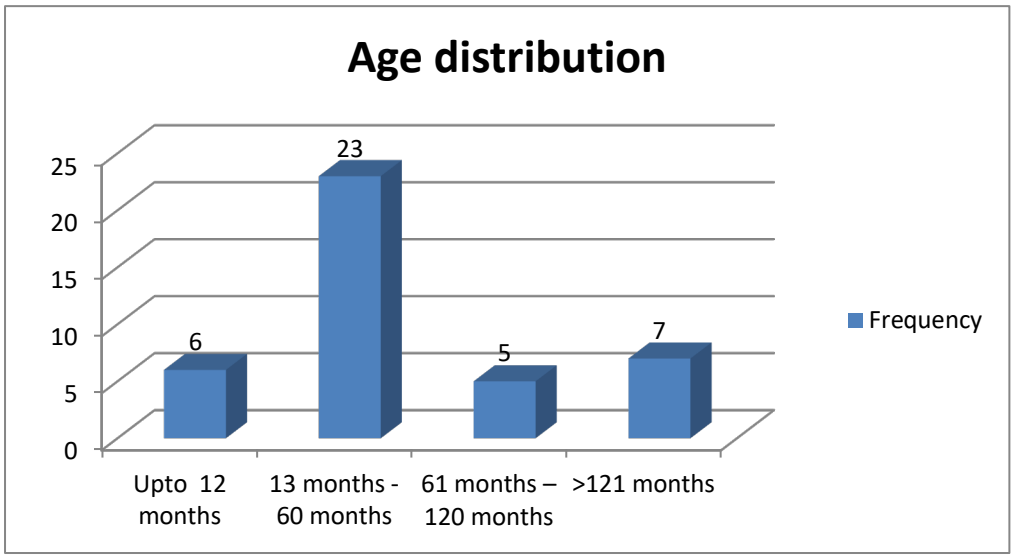
Study Design: A Comparative study. MRI/CT brain will be performed according to the clinical requirement and patient condition at initial admission. If there is clear diagnostic information on initial imaging no further neuroimaging will be performed on these children unless a prognostic imaging is deemed necessary by treating neurologist. Delayed imaging will be done when the initial neuroimaging is normal/nondiagnostic and child continues to have neurological symptoms and signs without obvious diagnoses from clinical, blood, CSF investigations. Also, when there are new onset neurological symptoms/ progressive worsening of neurological status after initial neuroimaging.

Results

Table 1: Distribution of age the patients

Age in months	Frequency	Percentage
Upto 12 months	6	14.6
13 months -60 months	23	56
61 months – 120 months	5	12.2
>121 months	7	17
Total	41	100

Maximum number of patients were in the age group of 13 to 60 months (56%) followed by 17% in >121 months age group and least number of patients were in 61 to 120 months age group (12.2%).



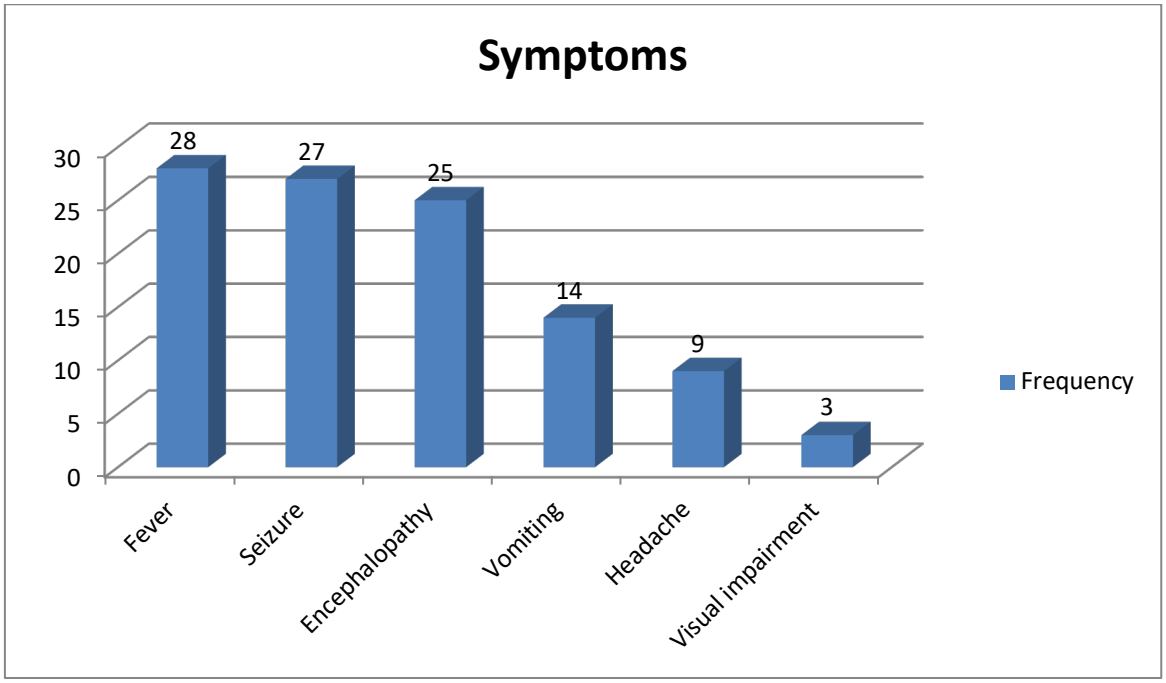
Graph 1: Distribution of age the patients

Table 2: Symptoms of the patients

Symptoms*	Frequency	Percentage
Fever	28	68.29
Seizure	27	65.85
Encephalopathy	25	60.9
Vomiting	14	34.14
Headache	9	21.95
Visual impairment	3	7.31

*More than one symptom can be present in a child.

Highest percentage of symptom was fever (68.29%) followed by seizure (65.85%) and encephalopathy (60.9%). Vomiting was in 34.14% of patients. Lowest percentage of symptom was Visual impairment. (21.95%).

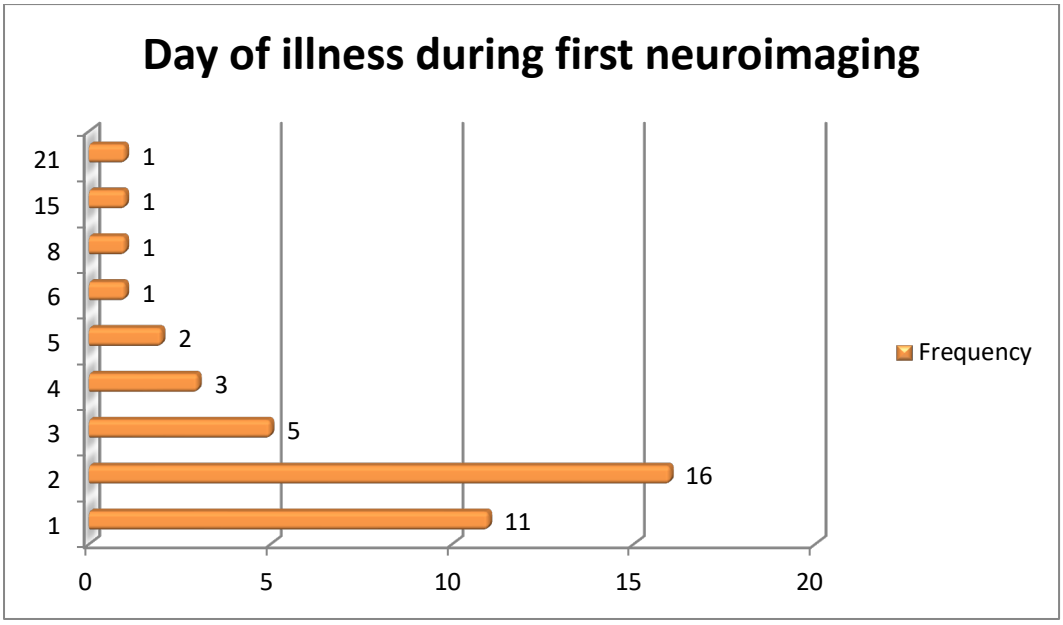


Graph 2: Symptoms of the patients

Table 3: Day of illness during first neuroimaging

Day of illness	Frequency	Percentage
1	11	26.82
2	16	39.02
3	5	12.19
4	3	7.31
5	2	4.87
6	1	2.43
8	1	2.43
15	1	2.43
21	1	2.43
Total	41	100

In 39.02 % of cases, first neuroimaging was done on Day-2 of illness and in 26.82% of cases, it was done on Day-1 of illness followed by 12.19% of patients on 3rd day of illness.

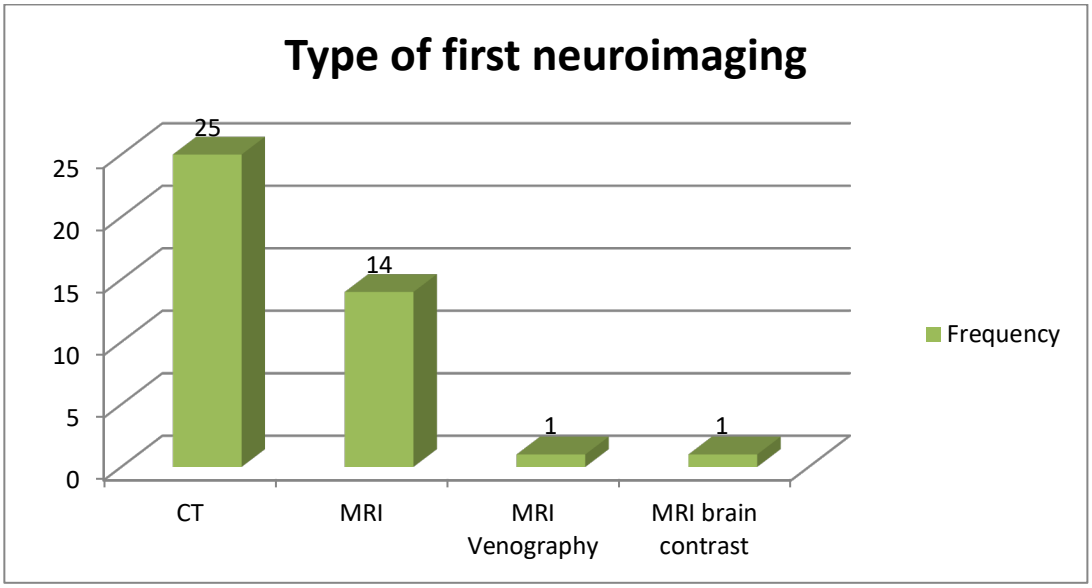


Graph 3: Day of illness during first neuroimaging

Table 4: First neuroimaging

Type of neuroimaging	Frequency	Percentage
CT head plain	25	60.97
MRI head plain	14	34.14
MRI brain Venography	1	2.43
MRI brain with contrast	1	2.43
Total	41	100

In 60.97% of cases, type of first neuroimaging in our study was CT head plain whereas in 34.14% of cases MRI head plain was done. MRI brain Venography and MRI brain with contrast was done in only 2.43% of cases.

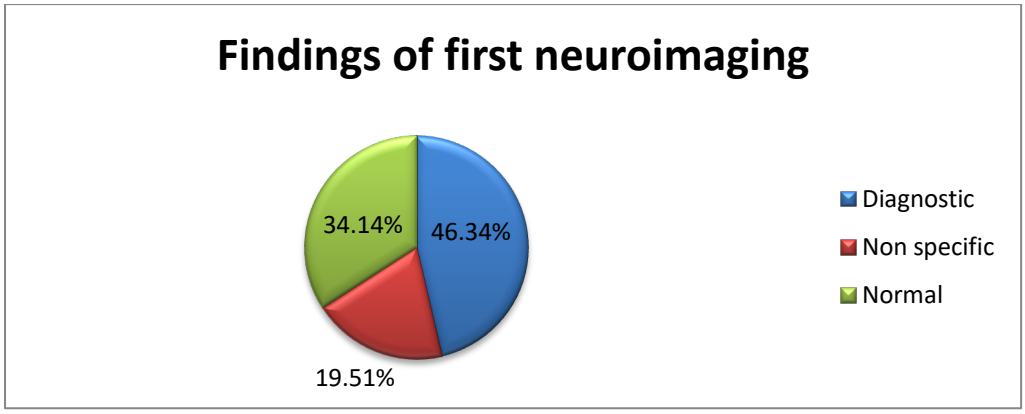


Graph 4: First neuroimaging

Table 5: Findings of first neuroimaging:

Finding	Frequency	Percentage
Diagnostic	19	46.34
Non specific	8	19.51
Normal	14	34.14
Total	41	100

In 46.34% of cases, first neuroimaging was diagnostic and in 34.14% of cases, it was normal and nonspecific findings were in 19.51% of cases.

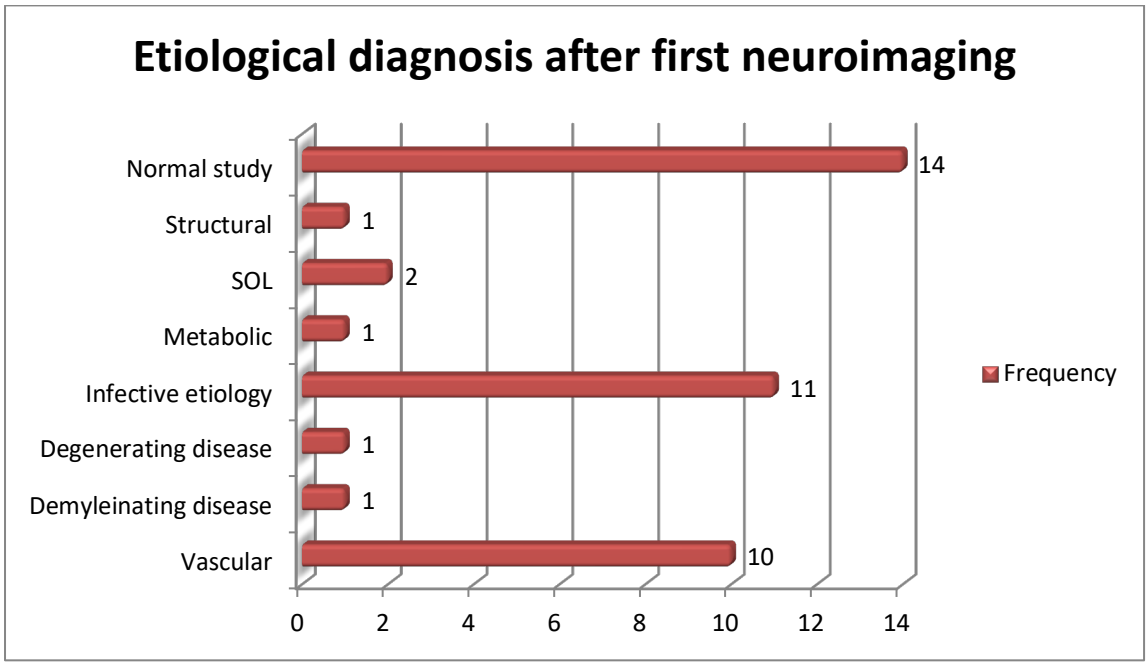


Graph 5: Findings of first neuroimaging

Table 6: Etiological diagnosis after 1st neuroimaging

Etiology	Frequency	Percentage
Vascular	10	24.3
Demyelinating disease	1	2.43
Degenerative disease	1	2.43
Infective etiology	11	26.82
Metabolic	1	2.43
SOL	2	4.86
Structural	1	2.43
Normal study	14	34.14
Total	41	100

In 34.14% of cases, first neuroimaging was normal whereas there was infective etiology in 26.82% of cases and vascular etiology in 24.3% of cases.

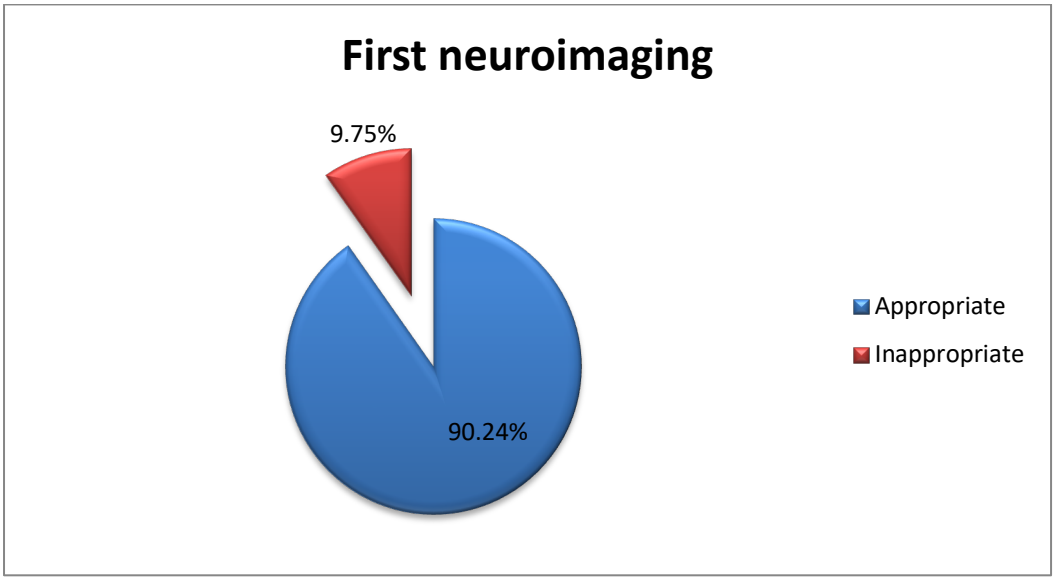


Graph 6: Etiological diagnosis after 1st neuroimaging:

Table 7: First neuroimaging:

First neuroimaging	Frequency	Percentage
Appropriate	37	90.24
Inappropriate	4	9.75
Total	41	100

In majority of cases (90.24%), first neuroimaging was Appropriate and was Inappropriate in only 9.75% of cases.

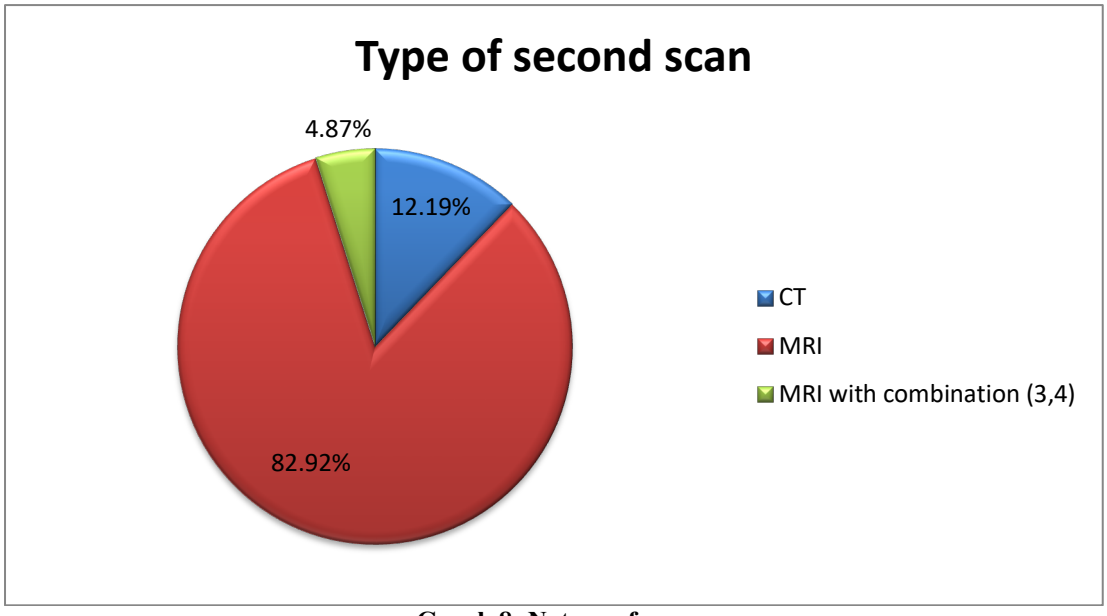


Graph 7: First neuroimaging

Table 8: Nature of scan

Type of scan	Frequency	Percentage
CT	5	12.19
MRI	34	82.92
MRI with combination (3,4)	2	4.87
Total	41	100

In majority of cases (82.92%), first neuroimaging was plain MRI head and CT scan was done in only 12.19%.

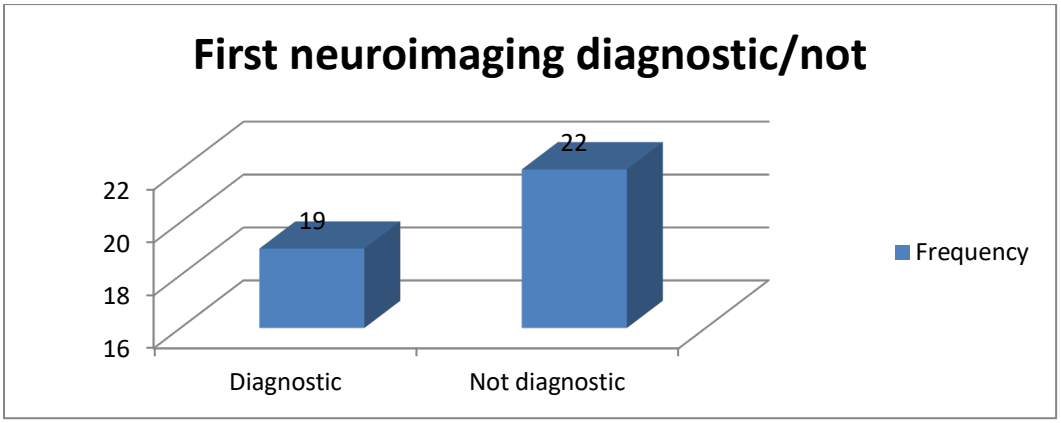


Graph 8: Nature of scan

Table 9: First neuroimaging diagnostic or not

First neuroimaging	Frequency	Percentage
Diagnostic	19	46.34
Not diagnostic	22	53.68
Total	41	100

First neuroimaging was diagnostic in 46.34% of cases and not diagnostic in 56.68%of cases.

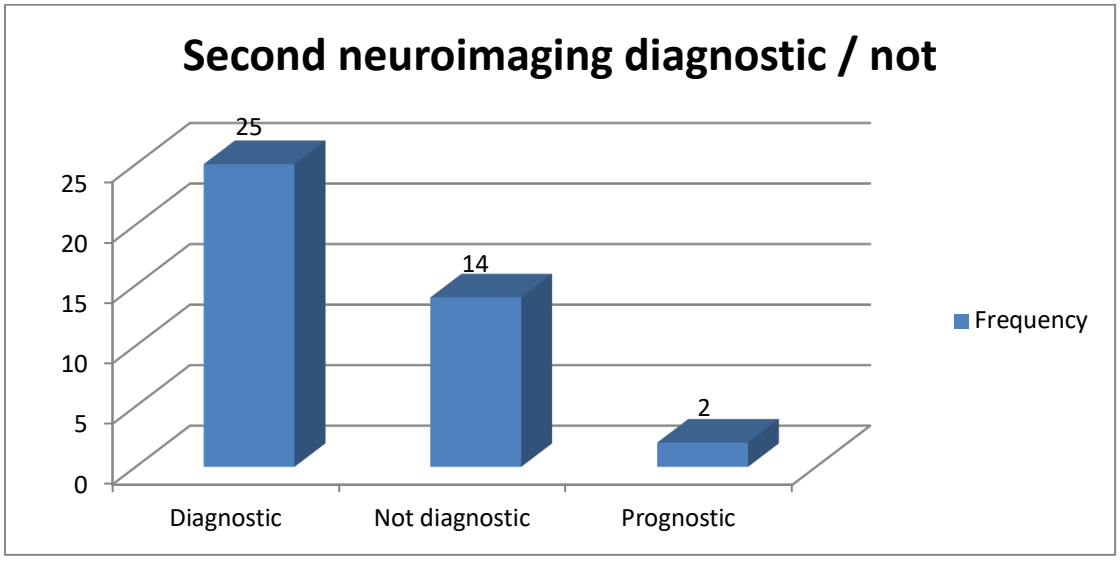


Graph 9: First neuroimaging diagnostic or not

Table 10: Second neuroimaging diagnostic or not

Second neuroimaging	Frequency	Percentage
Diagnostic	25	60.97
Not diagnostic	14	34.14
Prognostic	2	4.86
Total	41	100

Second neuroimaging was diagnostic in 60.97% of cases and nondiagnostic in 34.14% of cases.



Graph 10: Second neuroimaging diagnostic or not

Discussion

We identified a large cohort of children with Encephalopathy presenting symptoms. Encephalopathy describes a clinical syndrome of altered mental status, manifesting as reduced consciousness or altered behaviour. Encephalopathy may be caused by many diverse causes including, systemic infection, metabolic derangement, inherited metabolic disorders, toxins, hypoxia, trauma, vasculitis, and central nervous system infection. Neuroimaging: Only CT scan may be possible in the emergency situation but it may give valuable information such as presence of bleed, cerebral edema, temporal lobe hypodensities in herpes simplex encephalitis, thalamic abnormalities in JE, and basal exudates and hydrocephalus in tubercular meningitis. CT may also show brain herniation, effacement of cisterns, and infective collections such as brain abscesses and subdural empyema. If possible, an MRI should be obtained, as soon as the patient is stable. MRI is not needed if the etiology is clear by other investigations e.g., cerebral malaria, pyogenic meningitis; or if suggestive changes are seen on CT; or in epidemic situations where the likely etiology is already known. In all other patients, MRI provides useful information regarding the etiology and alternative diagnoses. However, the availability, cost, and difficulties in transporting sick and unstable patients for MRI may be limiting factors. MRI sequences should include diffusion weighted imaging to detect early changes, and a gadolinium enhanced study. In our study, maximum number of children suffering from NAE were present in the age group of 1 year to 5 years (56%) followed by 17% in >5 years of age and least num-

ber of children (12.6%) in 5-10 years age group. According to another study, while older age and longer seizure duration were associated with increased risk of urgent or emergent findings on neuroimaging. In that study, 4/11 children with these findings were under age 2 years. In our study, most common presenting symptomatology was Fever (68.29%) and second most common symptom was Seizure (65.85%). According to a study by Nishiyama M et al cases with viral etiologies had seizure, refusal of feeds, involuntary movements, GCS < 8, dystonia, and focal deficit as predominant clinical features. Similar clinical features were also observed in viral encephalitis cases by Karmakar et al.[1] Vomiting and meningeal signs were more prominent in pyogenic cases whereas CNS TB presented with headache, raised ICP, cranial nerve palsy, and dystonia. Meningeal signs were conspicuously absent in cerebral malaria.[2] According to one study, actual neuroimaging data was only available for 198 children (174 CT and 24 MRI) on another hand from six studies with a mean of 49% abnormal (range 29–70%). 31 However, these data were not limited to children with Encephalopathy. In a second study examining 144 children with Encephalopathy as a presentation of new-onset seizure, the combination of CT and MRI identified an underlying reason for SE in 30% of which 10% were described as "acute" abnormalities.[3] A recent analysis found that 11% of children with new unprovoked seizure had neuroimaging abnormalities but only 0.8% representing emergent findings. This study however only included 11% of patients with seizure > 15 minutes, and seizure duration > 15 minutes was associated with an increased incidence of abnormal neuroimaging findings.[4] In our study, first scan was done on second day in majority of cases (39%) and on first day in 27% of cases. Majority of children (78% i.e. 32/41) had their first scan within 3 days of neurologic symptom onset. First neuroimaging was CT scan plain in 60.97% of cases (25/41) and MRI head plain was done in 34% of cases (14/41). In our study, it was also found out that in 46.34% of cases (19/41), first neuroimaging was diagnostic and 34.14 (14/41) scans were normal. There were nonspecific findings in 19.51% of cases (8/41). Majority of normal scans were CT (11/14) and remaining 3 were MRI scans. In 8 scans, where findings were nonspecific, CT was done in 5 cases and remaining 3 were MRI. Etiological Diagnosis was analysed after first neuroimaging. Most common Etiological Diagnosis was Infective in 11/27 cases and second most common was Vascular Etiology in 10/27 cases. This could be explained by the great increase in the incidence of Acute Encephalopathy in the recent years in India, and the study population is belonging to a state with high burden for both the infections.[4,5] Recent studies also indicate an increasing prevalence of Acute Encephalopathy across the globe.[6] According to Kneen R et al only in about 60% of cases, etiology could be ascertained in his study. The probable reasons for such a big proportion of cases remaining etiologically inconclusive could be many, and includes: (i) specific serological and molecular-based investigations for many viral agents known to cause Acute Febrile Encephalopathy in children (e.g., human herpesvirus 6, West Nile virus, etc.) were not done; (ii) many cases of bacterial meningitis may have received over the counter antibiotics making the CSF bacteriologically sterile and difficult to interpret, and latex agglutination test for pneumococcus, Haemophilus influenzae or Neisseria meningitidis was not done in such cases; (iii) nonavailability of investigations for autoimmune encephalitis (e.g. CSF anti-N-methyl-D-aspartate receptor antibody, Anti-voltage gated potassium channel antibody, etc.).[7] However, again, in the earlier study by Kumar et al., even with extensive investigations for Acute Encephalopathy 40% cases remained etiologically unexplained.[3] The above-mentioned investigations are also not routinely available in most of the secondary and tertiary level health-care facilities of India and other developing countries, where such patients are being managed. The almost similar pattern of distribution of etiological diagnosis was in a recent study from central India.[8] Although the spectrum remains the same, the most common cause seems to vary according to the geographical region depending on the endemicity of different infective agents, with viral causes being predominant in Asia.[6,9] Our data add and expand to the previously reported yield of neuroimaging; in this cohort of children with Encephalopathy. In our study, majority of cases (82.92%) had Plain MRI Head (34/41) as second neuroimaging and only in 5 cases, CT was done.

Findings in both CT neuroimagings:

First neuroimaging	Second neuroimaging			Total
	CT	MRI	MRI with combination	
CT	2	23	0	25
MRI	2	10	2	14
MRI venogram	0	1	0	1
MRI contrast	1	0	0	1
Total	5	34	2	41

First and Secondneuroimagings were CT in 2 patients – Findings were same in both the neuroimagings. First and second neuroimagings were MRI in 10 patients – Findings were same in 2 cases, 8 cases had different findings. In the 8 cases, where there were different findings, management was changed. No intervention was done in 7 patients. From our study, it has been found out that most of the second neuroimagings (23/41-56.09%) were done from 4th-10th day of illness. Also second neuroimaging was diagnostic in 80.48% of cases. It clearly indicates that second neuroimaging was clearly beneficial. Reason for second neuroimaging was clinical worsening in 41.46% cases (17/41) and clinically children were not improved in 31.7% of cases (13/41). In majority of cases (78.04%-32/41), delayed neuroimaging and both neuroimagings were clearly beneficial in ANE. We found of 46.34% of children with abnormal neuroimaging of which 8.5% had pathology requiring urgent or emergent interventions. Practice guidelines regarding the need for emergent neuroimaging in status epilepticus are not conclusive. In 2006 the American Academy of Neurology published a practice parameter that was endorsed by the American Academy of Pediatrics recommending neuroimaging for the child with Encephalopathy "if there are clinical indications or if the etiology is unknown."[10] The Japanese Society of Child Neurology (JSCN) decided to establish clinical practice guidelines for Acute Encephalopathy call for neuroimaging for infants and children with recent onset Encephalopathy. While these guidelines are for children Recommendations. However, while none of the emergent abnormalities discovered on CT were missed by MRI, we recognize children with SE are frequently unstable and MRI may need to be deferred until clinical status has improved. Because CT scans can detect abnormalities in which earlier treatment may improve outcomes, CT can be a useful in unstable children in whom MRI cannot safely be performed in a reasonable time interval.

Conclusion

Consequently, paediatric neurologists may utilize several different tests at the same time to diagnose both the primary condition (the cause of encephalopathy) and the encephalopathy itself. This approach to diagnosis is done by most physicians, because encephalopathy is a complication that occurs because of a primary underlying health problem.

References

1. Studahl M. Influenza virus and CNS manifestations. J Clin Virol. 2003; 28:225-232.
2. Straussberg R, Harel L, Levy Y, et al. A syndrome of transient encephalopathy associated with adenovirus infection. Pedatrics. 2001; 107: e69.
3. Mastroyianni SD, Voudris KA, Katsarou E, et al. Acute necrotizing encephalopathy associated with parainfluenza virus in a Caucasian child. J Child Neurol. 2003; 18:570-572.
4. Takanashi J, Miyamoto T, Ando N, et al. Clinical and radiological features of rotavirus cerebellitis. AJNR Am J Neuroradiol. 2010;31: 1591-1595.
5. Rosas H, Wippold II FJ. West Nile Virus: Case report with MR imaging findings. AJNR Am J Neuroradiol. 2003; 24:1376-1378.
6. De Ory F, Avellón A, Echevarría JE, et al. Viral infections of the central nervous system in Spain: a prospective study. J Med Virol. 2013;85: 554-562.
7. Kamran S, Bener AB, Alper D, et al. Role of fluid-attenuated inversion recovery in the diagnosis of meningitis: comparison with contrast-enhanced magnetic resonance imaging. J Comput Assist Tomogr. 2004; 28:68-72.
8. Amin R, Ford-Jones E, Richardson SE, et al. Acute childhood encephalitis and encephalopathy associated with influenza. Pediatr Infect Dis. 2008; 27:390-395.
9. Glaser CA, Gilliam S, Schnurr D, et al. In search of encephalitis etiologies: Diagnostic challenges in the California Encephalitis Project, 1998d2000. Clin Infect Dis. 2003; 36:731-742.
10. Tunkel AR, Glaser CA, Bloch KC, et al. The management of encephalitis: Clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis. 2008; 47:303-327.