A DISSERTATION ON

CSF ANALYSIS IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE ENCEPHALITIS SYNDROME

M.D (BRANCH VII)

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CERTIFICATE

This is to certify that the dissertation titled **"CSF ANALYSIS IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE ENCEPHALITIS SYNDROME"** submitted by **Dr.S. DIVYA** to the Faculty of pediatrics, The Tamilnadu M.G.R.Medical University,Chennai in partial fulfillment of the requirement for the award of M.D.Degree (Pediatrics) is a bonafide research work carried out by her under our direct supervision and guidance.

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DECLARATION

I **Dr. S. DIVYA**, solemnly declare that the dissertation titled "**CSF ANALYSIS IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE ENCEPHALITIS SYNDROME"** has been prepared by me.

This is submitted to the **Tamilnadu Dr.M.G.R.Medical University**, Chennai in partial fulfillment of the rules and regulations for the M.D.Degree Examination in Paediatrics.

Place: Madurai

Date: **Dr. S. DIVYA**

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CONTENTS

INTRODUCTION

AES – Acute encephalitis syndrome.

Acute encephalitis syndrome in children includes a group of conditions which pose a diagnostic challenge to every pediatrician. Various conditions are included in the differential diagnosis of AES.

Definition of AES:[3]

AES includes any child presenting with fever, altered sensorium, seizures, symptoms of increased intracranial pressure (ICP).

AES accounts for about 10% to 15% of hospital admissions and is associated with significant morbidity and mortality.

Differential Diagnosis of AES:

1. Bacterial (pyogenic) meningitis
2. TB meningitis
3. Viral encephalitis
4. Aseptic meningitis
5. ADEM

6. Cerebral malaria
7. Reye's syndrome
8. CVT
9. Toxic / metabolic encephalopathies

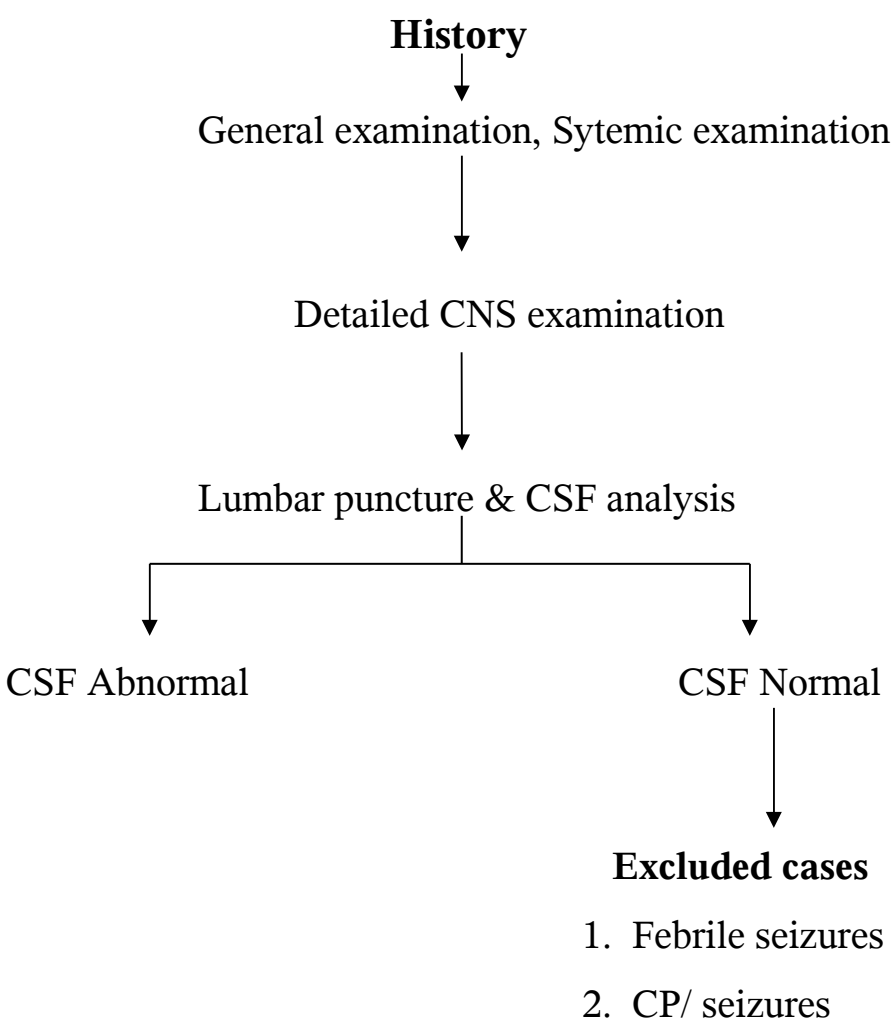
Lumbar puncture and CSF analysis helps in the differential diagnosis of AES. Clinical history, detailed systemic and neurological examination along with CSF findings provides the diagnostic clues towards the etiology of AES.

Neuroimaging studies helps in detecting the complications such as subdural effusion, infarcts, hydrocephalus ,etc in pyogenic and TB meningitis. Herpes viral encephalitis lesions are better picked up by MRI. MRI with gadolinium contrast remains the diagnostic imaging modality of choice in ADEM.

Prompt early diagnosis and management is necessary to avoid significant mortality and neurological impairment in children in AES.

This study was conducted to reemphasis the importance of CSF analysis in differential diagnosis of AES and in deciding the appropriate imaging modality of choice there by avoiding the unnecessary expenditure.

APPROACH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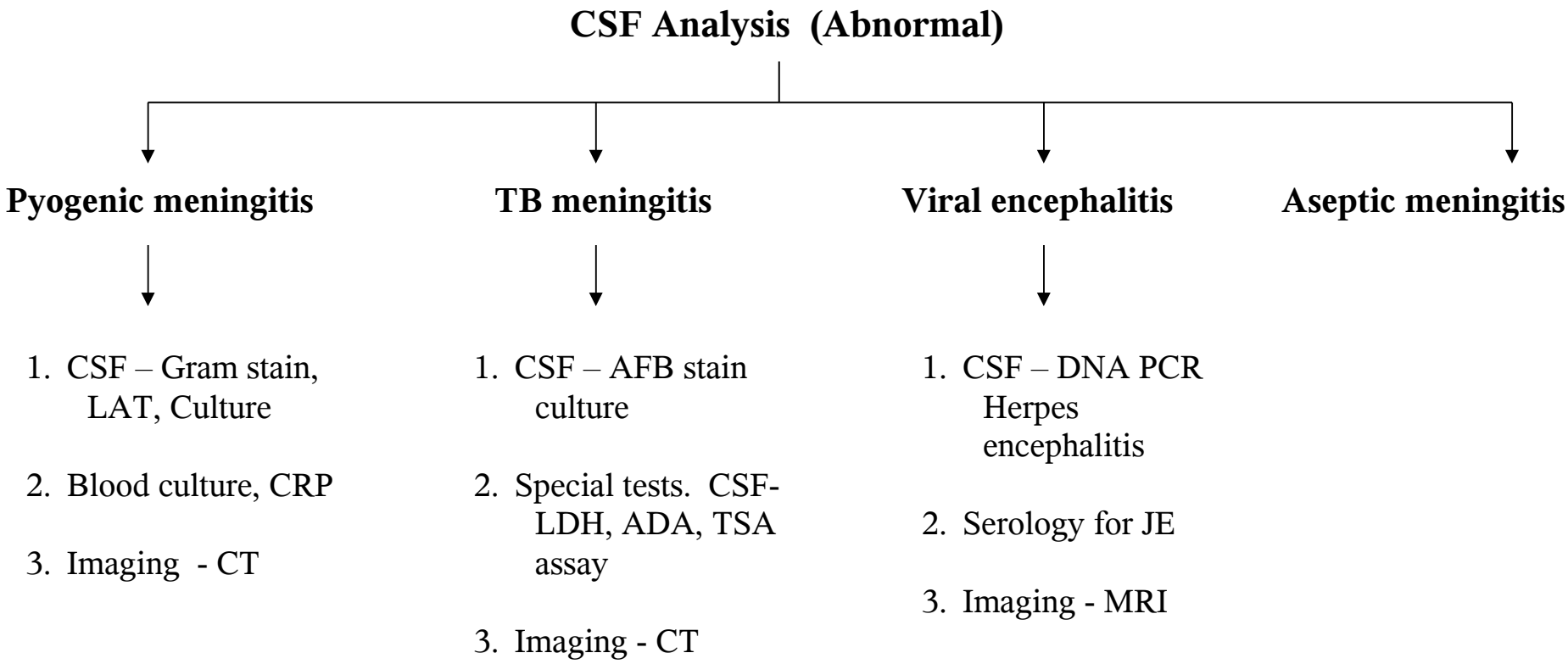
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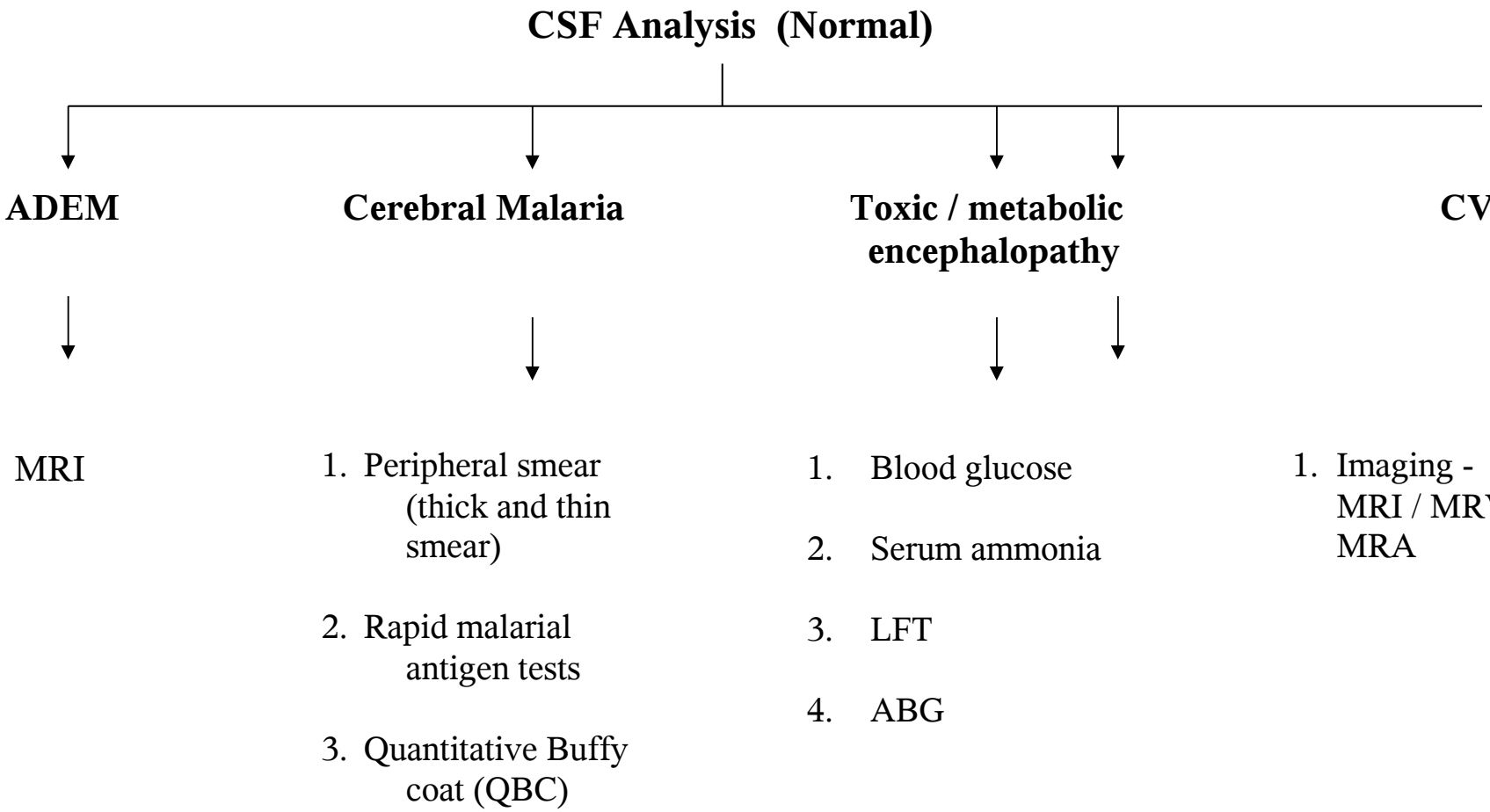
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3. LFT
4. ABG
1. Imaging - MRI / MRV / MRA

REVIEW OF LITERATURE

The importance of CSF analysis in particular the value of CSF cytology has been stressed in various literatures, as a preliminary step in the approval towards the diagnosis of acute infections of CNS.

Berry Schumann et al[1] stressed the importance of direct active involvement of the clinician in the evaluation of bedside investigation which had produced frequently more appropriate diagnosis.

Dyken[2] reports that counting chamber methods are useful quantitative procedures which helps to distinguish various types of cells in CSF.

In the words of Dougherty et al [4], CSF is obtained as an indirect assessment of brain status.

Quincke first described the techniques in LP way back in 1821,the single most important investigation in the diagnosis of CNS disease.

CSF cell count procedure is simple to perform with in a brief period does not requires sophisticated equipments at all times.

Scarbrough M, Thwaites G E,[5]reports that analysis of CSF is essential and helps in establishing the diagnosis of CNS disease especially in resource poor settings where the yield of diagnostic microbiology is low.

Neuman M,Tolford S,Harper M B,[6] reported the value of gram stain in the diagnosis of bacterial meningitis.

Oretega H W, Bonsu B K[7], analysed the importance of CSF pleocytosis along with biochemical tests in predicting bacterial meningitis when gram stain is negative or unavailable.

Bonsu B K[7], in their study on differentiating acute bacterial meningitis from acute viral meningitis among the children with CSF pleocytosis, observed that CSF neutrophils and CSF protein helps in differentiating the two entities.

Deivanayagan N, Ashok TP,[8] studied on the evaluation of CSF variables as a diagnostic test for bacterial meningitis observed that CSF polmorphonuclear pleocytosis along with low sugar and high protein values can be used as a diagnostic test for bacterial meningitis where positive culture rates are low .

Keith Morton [9]in his review article considered the total and differential cell count in CSF as standard tests.

CSF analysis being the most important in the diagnosis of CNS disease, clinician must be aware of the contraindications for LP.

Contraindications:[10]

1.Elevated intracranial pressure.

2.Symptoms and signs of impending cerebral herniation.

3.Skin infection at the site of LP.

4.Thrombocytopenia- platelet count below 20,000 cells / cu.mm.

5.Critically ill child (rare occasions).

Cerebrospinal fluid analysis:[10]

Normal opening pressure ranges from 50-80 mm H_20.In the setting of elevated intracranial pressure, it's usually above 100mmH_20.

Normal CSF is crystal clear. Cloudy CSF results from an increased cellularity. Normally CSF contains upto 5 WBC'S /mm^3. The presence of polymorphonuclear cells is always abnormal in a child. There are no RBC'S in normal CSF. Presence of red blood cells indicates either a traumatic tap or subarachnoid haemorrhage.

Gram stain is essential in the diagnosis of bacterial meningitis.AFB stain should be done if TBM is suspected. Other

special tests like Indian ink preparation is done only in special circumstances.

The CSF glucose concentration is about 60% of blood glucose levels. Protein values normally range from 10 to 40 mgs% in a child.

Latex agglutination test (LAT) helps in rapid detection of bacterial antigens in CSF. Polymerase chain reaction (PCR) in CSF has been a great advance in the diagnosis of Herpes encephalitis. CSF may also be tested for metabolites such as lactate, aminoacids, enolase levels in suspected metabolic diseases.

CSF FINDINGS IN CNS DISEASES[17]

Condition	Pressure (mmH2O)	Cell Count (mm3)	Protein (mg/dl)	Glucose (mg/dl)
Acute bacterial meningitis	100-300	100-10,000 or more, usually 200-2000 PMN's predominate	100 – 500	< 40 or < 2/3rd of blood glucose
TB meningitis	Usually elevated	10-500 ; PMN's early, but lymphocytes predominate later	100 – 3000 ; may be high in presence of block	< 50 in most cases
Partially treated bacterial meningitis	Normal or elevated	5-10,000 ; PMN usually	100 – 500	Normal or decreased
Viral meningitis or meningo encephalitis	Normal or slightly elevated (80-100)	Rarely > 1000 cells PMN's early later, mononuclear cells predominate	50 -200	Generally normal, may be decreased to < 40 in mumps (15-20%) of cases
Brain abscess	Usually elevated (100-300)	5 – 200, CSF may be acellular	75 – 500	Normal unless the abscess ruptures into ventricular system

TB MENINGITIS :

Tuberculosis of the central nervous system is the most serious complication of TB infection in children. CNS involvement will be present in 10% of children with TB infection .TB meningitis is the most serious and commonest form of neuro tuberculosis in children than tuberculoma . The highest incidence is between 9 months-9years of age group. In a Phillipine based study [12],they revealed that TBM in children usually arises as a complication of primary infection,usually with in 6 months of onset. Similar concept was also observed in Indian studies.[13] The important determinants for the development of TBM are age, poor nutritional status and associated HIV infection. There are three stages of TBM.

I – Prodromal stage.

II – Stage of meningeal irritation.

III- Stage of diffuse cerebral involvement.

Diagnosis of TBM

Early diagnosis of TBM is difficult due to gradual onset and vague symptomatology unless high index of suspicion is present.

The prodrome is often non specific with no one symptom predominating.

Illingworth[14] in his study reported 13% presented with fever,25% with vomiting,28% with headache and only 2% with meningitic symptoms.

In another study by Seth and Sharma,[15]clinical presentation were fever 100%, headache 80%,and neurological complications 50%.

Gold standard for the diagnosis of TBM is the isolation of the bacteria in the CSF. But bacilli are seldom seen by AFB staining and culture also takes several weeks. Children usually have paucibacillary type of TB which accounts for the low microbiological yield.

Poor response to treatment (or) morality in CNS TB is due to failure to begin the appropriate therapy in early stages. Hence various scoring systems are used to diagnose TBM. The most commonly used for the diagnosis of TBM in children is AIIMS criteria.

AIIMS criteria for diagnosis of TBM[16]

a) Essential criteria.

b) Supportive criteria.

Essential Criteria:

CSF showing

a) cells> 50cells /cu.mm predominant lymphocytes.

b)Proteins > 60 mg%.

c)Sugar <2/3rd of blood sugar.

Supportive Criteria:

a) H/o fever of 2 wks or more.

b) H/o contact with TB.

c) Mantoux positivity.

d) Radiological evidence of TB elsewhere in the body.

e) Generalised lymphadenopathy.

f) CT scan evidence – basal exudates, Hydrocephalus, infarcts, gyral enhancement.

g) Isolation of AFB from gastric lavage.

h) Histologically proven TB adenitis.

Definitive diagnosis is by the isolation of AFB in CSF.

Probable TBM - Essential criteria + atleast 2 supportive criteria.

Neuroimaging studies have greatly enhanced the diagnostic accuracy of neurotuberculosis, but still not pathognomonic for the disease. Common CT findings includes basilar exudates, ventricular dilatation, infarcts and intracranial granuloma.

CSF seldom reveals the acid fast bacilli (AFB), there is an need for rapid diagnostic tests for the confirmation of TB. Many newer methods have been developed to establish an early and definitive diagnosis of TBM such as ADA assay, Bromide partition test, LDH levels, TSA assay and immunological tests which detects the mycobacterial antigen or antimycobacterial antibody in CSF, but no single test with adequate sensitivity and specificity is available at present.

Prognosis in CNS TB depends upon early diagnosis and institution of appropriate therapy.

Bacterial meningitis:

Bacterial meningitis is an important potentially serious infection in infants and in older children. Despite the advances in vaccination and antibiotics , bacterial meningitis remains a major cause of death and long term neurological disabilities such as mental retardation, seizures etc. Hence early diagnosis is a must. Bacterial

meningitis should be considered in the differential diagnosis of highly febrile children with altered mental status and other evidence of neurological dysfunction.[17]

The etiological agents vary depending upon the age group.

< 2 months :

Group B streptococci, Listeria monocytogenes, E .Coli

2 months – 12 yrs:

Streptococcal pneumoniae, haemophilus influenza and Neisseria meningitis.

Haemophilus influenza remains the most common etiological agent in children between 2months – 2 yrs of age particularly in developing countries due to lack of vaccination against H. influenza.

Most of the cases of pyogenic meningitis occurs in < 1 yr of age group.Bandaru rao et al [18] in a study on acute bacterial meningitis,reported that 65% of cases were <1 year age group.

Chandramuki et al [19] in the study on bacteriological profile on community acquired acute bacterial meningitis, reported gram stain remains a simple, rapid, inexpensive means of diagnosing the organisms.

Bacterial meningitis most commonly results from haematogeneous dissemination of micro organisms from a distant site of infection. Bacteremia will be present and the blood cultures detects the organisms in 50% of patients.[17] The diagnosis of bacterial meningitis is confirmed by CSF examination showing polymorpho nuclear (pus) cells. Microbiological lab plays a critical role in early identification of casuative organisms. Gram stain positivity varies from 70-90%. Latex agglutination test (LAT) which is a rapid diagnostic test which detects the antigens against H. influenza S.pneumonia, N. Meningitidis, group B streptococci.

LAT is particularly useful in identifying the organisms when antibiotics are used prior to CSF examination where Gram stain and culture becomes negative.But latex test will not be of use in detecting the gram negative organisms.

The role of neuroimaging is to evaluate the complications such as abscess, ventriculitis and subdural effusion.

Kumar et el[20] in the study on value of CT scan in the diagnosis of meningitis, reported that subdural effusion was more common in pyogenic meningitis.

VIRAL ENCEPHALITIS

Viral encephalitis is an acute inflammatory process involving the meninges and the brain parenchyma. The most common etiological agent for viral encephalitis are the enteroviruses. Herpes simplex viruses (HSV-1) is an important cause of severe sporadic encephalitis. Brain involvement is usually focal particularly the temporal lobe which is most commonly affected. Arbovirus encephalitis usually occurs in epidemics. Japanese Encephalitis (JE) is the most common Arboviral encephalitis in south east Asia.

Neurological damage is caused by direct invasion of neural tissue by virus (or) by host reaction to viral antigens. Some of the viruses will have predilection towards certain areas of brain. Rabies virus have predilection for basal structures and JE virus towards basal ganglia and thalami.

Diagnosis of viral encephalitis is usually made on the basis of clinical presentation along with CSF findings. Detection of viral DNA by Polymerase chain Reaction (PCR) is particularly useful in detecting HSV which is a potentially treatable condition among the viral encephalitis. Serology for JE antibodies may be of use in detecting JE viral encephalitis.

Neuroimaging plays an important role in diagnosis of viral encephalitis. MRI is the imaging modality of choice in HSV. Typical EEG abnormalities PLED'S 2-3 Hz from temporal lobe every 2-3 seconds are noted in 80% of cases of HSV.

Dengue infection is one of the known cause of acute febrile encephalopathy in children.

Chitsanu pancharoen et el[21] described the neurological manifestations in dengue patients. Dengue virus is a non – neurotropic virus. The neurological complications in dengue infection may be due to intracranial bleed, cerebral edema,hepatic encephalopathy etc., Encephalitis due to dengue virus is one of the rare and atypical presentations of dengue in children.

HIV :

HIV encephalopathy is a known clinical entity. CNS involvement in HIV infection is more common in children than in adults. Macrophages and microglia play an important role in HIV neuropathogenesis. The developing brain in young infants may be affected directly by the virus itself which infects the various brain cells or virus may cause indirect damage by the release of cytokines such as IL-1 α, 1β, TNF- α , IL-2.

Vardhaman S. Udgirkar et al[50] in his article on the neurological manifestations of HIV infection reports that HIV encephalopathy can be a presenting manifestation among the infected children. The risk of HIV encephalopathy is also correlated directly with the severity of HIV related symptoms, CD4 cell count and P24 antigen levels in the mother.

HIV infection should be suspected in children presenting with unexplained neurological manifestations and growth failure.

Pediatricians will have to be alert to the possibility of HIV as an etiological agent in infants and children presenting with mental subnormality and unexplained neurological deficit.

ADEM:

Acute disseminated encephalomyelitis (ADEM) is a monophasic acute inflammatory demyelinating disorder of CNS. It is an immune mediated inflammatory process following viral infection (or) vaccination. Viral pathogens commonly implicated are measles, varicella, HSV, HIV, EBV, HepB, rubella, coxsackie virus.

The hallmark of the disease is acute development of neurological signs accompanied by fever, headache and altered sensorium with

focal neurological signs. ADEM can mimic many other neurological diseases especially meningo encephalitis.

There are two clinical forms of presentation in ADEM. Encephalitic and myelitic form. In some cases, haemorrhagic forms have also been reported.[22]

In a case series on ADEM, it was reported that ADEM presents with symptoms and signs of meningoencephalitis.[23]

CSF analysis being normal (or) showing mild elevation of protein and mild lymphocytic pleocytosis, the diagnosis of ADEM is by neuroimaging especially MRI with gadolinium contrast. Screening of the spinal cord is also necessary to detect the lesions in the cord.

MRI reveals the characteristic lesions involving subcortical white matter and deep seated gray matter including basal ganglia, thalamus and cerebellum, in T2 weighted images.

CT scan may be normal in ADEM or reveals the areas of patchy low attenuation in the white matter[24].

MRI should be considered early in patients with acute onset of unexplained encephalopathy with or without focal neurological deficit particularly when the CSF analysis is normal.

Reye's Syndrome :

Reye's syndrome is characterized by acute non inflammatory encephalopathy and hepatic failure. It is more common in children. The exact etiology remains unknown but it is precipitated by a viral infection especially influenza, varicella and the use of salicylates during the illness.

Reye's syndrome should be considered in the differential diagnosis in any child with vomiting and altered mental status.[25] It results from mitochondrial dysfunction that inhibits oxidative phosphorylation and fatty acid beta oxidation.

Dr.D.Ghosh et el[26] in his study on the investigation of Reye's syndrome described the cases presented with the symptoms of meningoencephalitis. He reported that history of fever was present in 83%, vomiting preceding unconsciousness in 83% cases, abnormal posturing in 55%, abnormal behavior in 65% cases.

T. Jacob John in his article [51] on the outbreaks of killer brain disease in children, reports that Reye's syndrome can also occur in outbreaks similar to the outbreak of viral (JE) encephalitis.

Unless a high index of suspicion is present most of the cases will be erroneously misdiagnosed as encephalitis without performing the necessary investigations for the diagnosis of Reye's syndrome.

IEM that may mimic Reye's syndrome include fatty acid oxidation defects, amino and organic acidopathies and urea cycle defects.

The clinical features of reye'syndrome are described in four stages:

Stage I : vomiting,anorexia,mild confusion,listlessness,apathy.

StageII : delirium, restlessness, irritability, lack of orientation, agitated states.

Stage III : coma, decorticate posture which later becomes decerebrate, patients may die.

Stage IV: flaccidity, areflexia, apnea, pupils not reacting to light, hypotension.

CSF analysis being normal the other lab tests that favours the diagnosis are elevated serum ammonia level, elevated prothrombin time, coagulation abnormalities, elevated transaminases and normal bilirubin levels. Neuroimaging is usually normal some times may show cerebral edema. A high index of suspicion is necessary for the diagnosis of Reye's syndrome.

Cerebral Malaria :

Malaria is an important cause of morbidity and mortality in South east Asia. 50% of these cases are due to infection with plasmodium falciparum. Cerebral malaria is one of the dreadful complication of P.falciparum infection.Clinical presentation includes fever, seizures and altered mental status or unconsiousness which is easily confused with diagnosis of AES .

There are case reports of cerebral malaria mimicking meningoencephalitis with meningeal signs.[27]

Cerebral malaria is due to blockage and sequestration of capillaries with parasitized RBC's. CSF analysis is normal usually and imaging studies is often normal or shows cerebral edema. The gold standard test for the diagnosis of malaria is by identification of malarial parasite by peripheral smear both thick and thin smear examinations.

Quantitative Buffy Coat :

QBC test is a new method for the identification of the malarial parasite in the peripheral blood.

In this test, blood sample is centrifuged and RBC's are stained with acridine orange and is examined under UV light source. QBC is fast, easy and more sensitive than thick smear examination.

Rapid Diagnostic Tests :[52]

Rapid tests detect the malarial antigens such as pf HRP2 / PMA / PLDH using the monoclonal antibodies. The presence of parasite antigen in blood is detected by colour changes on the strip test. Rapid tests are quick and easy to perform but they are only qualitative tests not useful in prognostication of the disease.

Cerebral malaria unless diagnosed early and treated appropriately will have a poor prognosis. It should be considered in dd of AES particularly case from an endemic area, presence of pallor, splenomegaly along with altered level of consciousness.

CVT :

Cerebral venous thrombosis (CVT) is an important cause of stroke in children but is less common than other types of strokes. But it is more challenging to diagnose. It results from thrombosis of the dural venous sinuses which drains the blood from the brain.

Superior saggital sinus and lateral sinuses are the most commonly involved sinuses. Signs and symptoms includes fever,

headache, seizures, altered mental status due to increased ICP, focal neurological deficit. It can present as acute encephalopathy. Neuroimaging studies forms the main stay of diagnosis of CVT.

IEM :

Inborn error of metabolism are conditions caused by genetic defects related to synthesis, metabolism, transport or storage of biochemical compounds. It usually result from deficiency of one or more enzymes.

IEM presents as acute encephalopathy or chronic progressive encephalopathy. IEM presenting as acute encephalopathy are organic acidemias, urea cycle disorders, fatty acid oxidation defects and mitochondrial disorders.[28] Basic lab work up in these cases includes blood glucose, serum ammonia, ABG and serum lactate levels.

IEM mimics many of the common pediatric illness such as sepsis.IEM should be considered in the differential diagnosis of any sick neonate presenting with the clinical picture of sepsis,HIE, congenital infections,duct dependant cardiac lesions.[29]

Prompt detection requires a high index of suspicion and the early measurement of biochemical markers such as serum

ammonia.Diagnosis is important not only for treatment but also for the genetic counselling.

Neuroimaging studies in IEM may provide helpful pointers towards etiology in some cases where IEM may be associated with structural malformations.[29]

In glutaric aciduria,frontotemporal atrophy may be noted.Zellweger syndrome is associated with diffuse cortical migration abnormalities.

Similarly EEG abnormalities may be suggestive of particular IEM eg:burst suppression pattern in non –ketotic hyperglycinemia,comb- like rhythm in MSUD.

AIM OF THE STUDY

1. To reemphasis the importance of CSF analysis in the differential diagnosis of AES.
2. To correlate the CSF findings with neuroimaging studies.

MATERIALS AND METHODS

Study Centre :

The study was conducted in the Institute of Child Health and Research Centre, Government Rajaji Hospital, Madurai Medical College, Madurai.

Study Period :

The study was carried out prospectively from December 2007 to May 2009.

Study Design :

Prospective Observational Study.

Study Population :

Children admitted in Govt. Rajaji Hospital, Madurai Medical College, Madurai.

Sample size :

111 cases.

Inclusion Criteria :

Children between 2 months to 12 years with a clinical diagnosis of AES.

Exclusion Criteria :

1. Age < 2 months and > 12 years.
2. Repeat lumbar puncture.

Conflict of interest : Nil

Financial Support : Nil

Ethical Committee clearance : Obtained.

Methodology :

For all children admitted with clinical picture of AES, a detailed history, general examination, systemic examination and detailed neurological examination were made. Informed written consent was obtained from the parents for the procedure of lumbar puncture. Lumbar puncture was done in all cases unless absolute contraindications for LP such as skin infection at the site of the LP, thrombocytopenia, increased ICP with signs of pending cerebral herniation were present. CSF analysis was done.

For all the cases, CSF cytology was done by a single observer who is the principal investigator in the study for better results. Separate consent for doing cell count by the investigator was not obtained as it is a part of service provided. CSF cell count was done

by using the Fuch's Rosenthal chamber with in half an hour of collecting the sample.

CSF cell count technique : [54]

Uncentrifuged CSF is diluted 1 in 2 by mixing 1 drop of CSF with 1 drop of diluting fluid using fine bore pipettes so that drops will be of equal volume. Using the capillary tube, Fuch's Rosenthal chamber is filled with this diluted CSF carefully so that CSF does not overflow into the channels on each side of the chamber. Counting chamber and its rulings are focused and cells are counted in 5 large squares and multiplied by 2 to give the total count per cumm. Differential count done by centrifuging the CSF sample, and by staining with methylene blue.

Biochemical analysis of CSF protein, glucose, chloride and globulin were done. Simultaneous estimation of blood glucose levels were done to avoid spuriously elevated blood / CSF glucose ratio. Microbiological work up including CSF gram stain, Latex agglutination test, CSF culture, blood culture and CRP was done in cases where the CSF analysis favours pyogenic meningitis. AFB stain in CSF, mantoux test, chest x ray, Gastric juice for AFB was done in cases suggestive of TB meningitis. Serology for JE was done

in cases with viral encephalitis. All cases were subjected to neuroimaging irrespective of CSF analysis.

All children were effectively managed with appropriate measures along with nursing care. Children who developed complication like hydrocephalus were referred to neurosurgery department for surgical management.

Statistical Methods :

Data collected were recorded in a Master chart. Data analysis was done with the help of computer using SSPS software. Data was analysed using simple descriptive statistics.

RESULTS AND ANALYSIS

In this study, 111 cases with clinical picture suggestive of AES were studied.

Age group distribution

Table 1

Age group	No. of cases	%
<1yr	29	26.1
1 to <3yrs	26	23.4
3 to <5yrs	18	16.2
5 to <8yrs	26	23.4
>8yrs	12	10.9
Total	111	100

29 cases were <1yr of age, 26 cases were between 1 to <3yrs. 49.5% cases were in the age group of less than 3yrs.

Sex distribution

Table 2

Sex	Cases No.	Cases %
Male	57	51.4
Female	54	48.6
Total	111	100

Out of 111 cases 57 (51.4%) were males and 54 (48.6%) were females.

Symptomatology

Table 3

Symptoms	Yes	%	No	%
Fever	111	100	-	-
Altered sensorium	91	82	20	18
Seizures	61	55	50	45

In this study, among the 111 cases, fever was present in 111 (100%), Altered sensorium in 91 (82%), seizures in 61 (55%) of cases.

With Status epilepticus

Table 4

Status epilepticus	Cases	
	No.	%
Yes	18	29.5
No	43	70.5
Total	61	100

In this study, out of 61 cases with seizures 18 (29.5%) had status epilepticus.

With meningeal signs.

Table 5

Meningeal Signs	Cases	
	No.	%
Yes	41	51.2
No	39	48.8

In this study, 41 (51.2%) cases had signs of meningeal irritation while in 39 (48.8%) cases signs were absent.

CSF cytology

Table 6

CSF cytology	Cases	
	No.	%
Normal	55	49.5
Abnormal	56	50.5
Total	111	100

In this study out of 111 cases, 55(49.5%) cases had normal CSF cytology and 56 (50.5%) cases had abnormal cytology.

Table 7

Abnormal CSF Cytology

DIAGNOSIS	Cases	
	No.	%
Pyogenic meningitis	21	37.5
TBM	21	37.5
Aseptic meningitis	7	12.5
Viral encephalitis	4	7.1
ADEM	3	5.4
Total	56	100

Out of 56 cases with abnormal CSF cytology, Pyogenic meningitis accounts for 21 (37.5%), Aseptic Meningitis 7(12.5%) cases, TBM 21 (37.5%) cases, viral encephalitis 4(7.1%) and ADEM 3(5.4%) cases.

Normal CSF cytology

Table 8

Diagnosis	Cases	
	No.	%
ADEM	6	10.4
Dengue encephalopathy	2	3.6
Acute encephalopathy	5	9.0
Post meningitic sequelae	5	9.0
Cerebral malaria	1	1.8
Brain abscess	1	1.8
Drug toxicity	1	1.8
Viral encephalitis	6	10.9
CP/ Seizures	5	9.0
Neem oil encephalopathy	2	3.6
Febrile seizures	19	34.54
HIV	2	3.6
Total	55	100

Out of 55 cases with abnormal cytology, ADEM accounts for 6 (10.9%) cases, febrile seizures 19 (34.54%), Acute encephalopathy and post meningitic sequelae each accounts for 5 (9%) cases.

Pyogenic Meningitis

Table 9

Age wise distribution

Age group	Cases	
	No.	%
<1 yr	13	61.9
1 to < 3yrs	6	28.6
3 to <5yrs	1	4.8
5to <8 Yrs	1	4.8
Total	21	100

Out of 21 cases of pyogenic meningitis, 13 (61.9%) cases were <1yr of age.

Causative organisms in pyogenic meningitis

Table 10

Organisms	Cases	
	No.	%
H. influenza	10	47.6
S. pneumoniae	2	9.5
Group B streptococci	1	4.8
Kleibsiella	1	4.8
Gram +ive cocci	2	9.5
Not identified	5	23.8
Total	21	100

Out of 21 cases, organisms were identified in 16 (76.2%) of cases. H. influenza was the most common organism isolated .

Table 11

VARIOUS METHODS OF IDENTIFYING THE ORGANISMS

ORGANISMS	No.	Gram stain (I)	Latex (II)	Culture (III)
H . influenza	10	7	10	-
S.Pneumonia	2	1	2	1
Group B streptococci	1	1	1	1
Kleibsiella	1	-	-	1
Gram positive cocci	2	2	-	-
Not identified	5	-	-	-
Total	21	11	13	3
P Value	II & III - 0.004 I & III - 0.02			

Out of 21cases, Gram Stain was positive in 11 (52.45%) cases, Latex was positive in 13(61.9%)cases, culture in 3(14.3%) cases.

Latex and gram stain is better in identifying the organisms than culture ($p = <0.05$).

Table 12

Biochemical analysis in pyogenic meningitis.

Glucose	Protein		Total
	Normal	High	
Low	1	9	10
Normal	2	9	11
Total	3	18	21
P Value	p = 0.02		

Among the 21 cases of pyogenic Meningitis, high protein values were observed in 18 (85.7%) cases, low glucose in 10(47.6%) cases and low glucose & high protein 9(42.9%) cases .

Protein values have a better diagnostic value than glucose levels (p=0.02).

Table 13

Imaging in pyogenic meningitis

Findings	Cases	
	No.	%
Leptomeningeal enhancement	15	71.4
B/l Subdural hygroma	1	4.8
Infarct	1	4.8
Normal	1	4.8
Not taken	3	14.3
Total	21	100

In this study, out of 21 cases of pyogenic meningitis Leptomeningeal enhancement was observed in 15(71.4%) cases in the neuroimaging (CT).

TB Meningitis

Table 14

Age wise distribution of TB Meningitis

Age Group	Cases	
	No.	%
< 1Yrs	2	9.5
1 to<3 yrs	3	14.3
3 to < 5Yrs	4	19.0
5 to 8 Yrs	11	52.4
<8 Yrs	1	4.8
Total	21	100

In this study, the incidence of TBM was highest in 3 to 8 yrs of age (71.4%).

Table - 15

Clinical Findings in TBM

Findings	Yes	No
I) Contact h/o	12(57.1%)	9(42.9)%
II)BCG Scar	13 (61.9%)	8 (38.1%)
III)Mantoux	1(4.8)%	20(95.2)%
IV)Chest x-ray abnormal	-	21(100)%
P Value between I & II	1.0 (Not significant)	

In this study, contact H/o was positive in 12(57.1%) cases, BCG Scar was present in 13(61.9%) cases. Mantoux positivity in 1 (4.8%) case.

These is no significant difference between those with BCG scar and those without scar (p value = 1.0).

Table 16

Biochemical analysis in TB meningitis.

Glucose	Protein		Total
	Normal	High	
Low	3	6	42.9%
Normal	2	10	52.1%
Total	23.8%	76.2%	100%

In this study, high protein values was observed in 16(76.2%) cases. Protein values significantly correlate than glucose values (P value = 0.03).

Table 17

Imaging in TB meningitis

Findings	Cases	
	No.	%
Leptomeningeal enhancement	7	33.3
Hydrocephalus	9	42.9
Cerebral edema	1	4.8
Granulomatous lesion	1	4.8
Hypodense lesion	1	4.8
Normal	2	9.5
Total	21	100

In this study, hydrocephalus was the most common finding observed in 9(42.9%) cases.

Viral encephalitis

Table – 18.

Imaging in viral encephalitis

CT Scan Findings	Cases	
	No.	%
Leptomeningeal enhancement	3	30
Cerebral edema	4	40
Hypodense lesion	1	10
B/L Subdural hygroma	1	10
Normal	1	10

In this study, abnormal findings were reported in 9 out of 10 cases in CT Scan.

Table –19

Comparison of CT Vs MRI in viral encephalitis

CT Scan findings	**MRI**	
Lepto meningeal enhancement(3)	Herpes (1)	Not taken(2)
Cerebral edema (4)	Herpes(2)	Non Specific viral Meningitis (2)
Normal (1)	Herpes (1)	---
Hypodense lesion (1)	Herpes (1)	
B/L Subdural hygroma	---	Non-specific viral

In this study, it was observed that MRI is the better imaging modality of choice in detecting Herpes Viral encephalitis (p value = 0.02).

Table – 20.

Serology in viral encephalitis

CSF JE Antibodies	Cases	
	No.	%
Positive	3	30
Negative	7	70

In this study, 3 cases were JE positive in CSF samples.

Table – 21

Imaging in the CSF normal cytology group

Findings	Imaging	
	CT	MRI
Normal	10 (34.5%)	6 (22.2%)
Abnormal	19 (65.5%)	22 (77.8%)

In this study, out of 29 cases with CSF normal study imaging MRI was abnormal in 22 (77.8%) cases, CT was abnormal in 19(65.5%) cases.

DISCUSSION

Acute encephalitis syndrome (AES) includes a list of conditions with a similar clinical presentation posing a great diagnostic challenge to every pediatrician. Correct diagnosis and early institution of appropriate therapy is necessary to avoid significant morbidity and mortality and also to avoid polypharmacy. CSF analysis helps in the differential diagnosis of AES to a great extent. The importance of CSF cell count analysis in the differential diagnosis of AES was reemphasized in this study.

This study included 111 children in the age group of 2 months – 12 years with a clinical picture suggestive of AES.

In this study, 57 (51.4%) were males and 54 (48.6%) were females.

54 (49.5%) were less than 3 years of age, 18 (16.2%) were in the age group between 3 to < 5 yrs, 26 (23.4%) were in the age group between 5 to < 8 years and 12 (10.8%) were > 8 years.

In the clinical presentation, Fever was present in 111(100%) cases, altered sensorium in 91 (82%) and seizures in 61 (55%).

Out of 61 cases with seizures, 18 (29.5%) cases had status epilepticus. Febrile seizures was the most common cause of status epilepticus in this study.

In this study,41(51.3%) cases had signs of meningeal irritation where in 39(48.8%) cases the signs were absent.

Meningeal signs were applicable to only those children with AF closed. Out of 111 cases , 80 cases falls into this category.

In this study, 55 (49.5%) cases had normal CSF cytology and 56 (50.5%) had abnormal CSF cytology.

PYOGENIC MENINGITIS :

In this study, among the 21 cases of pyogenic meningitis, 15 (61.9%) cases were in the age group of < 1 yr which is the most vulnerable age group for bacterial meningitis.

Bandaru Rao et al [18], in their study on etiology and clinical profile of acute bacterial meningitis reported that 65% of cases were < 1 yr of age which correlates well with this study.

In a study by F. Clifford Rose[30] on the diagnosis of pyogenic meningitis, low glucose values in CSF was observed in 70% and raised protein levels in 90% of cases .

In this study, raised protein levels in CSF was observed in 85.7% and low glucose values in 47.6% cases. Raised protein values have a better diagnostic value than low glucose levels in this study.

Causative organisms identified by combining Gramstain and or latex agglutination and or culture of CSF were in 16 (76.2%) cases.

Among the identified etiological agents, H.influenza accounts for 10 (62.5%) cases, S.pneumonia 2 (12.5%) cases,group B streptococci and kleibsiella each (6.5%) cases respectively.

In our study, Gram stain provided an evidence of the etiological agents in 11 (52.45%) cases which correlated with various other studies[18,19] reporting the sensitivity of gram stain between 60-90%.

Chandramukhi A et al [19] in their study on the bacteriological profile of community acquired acute bacterial meningitis reported the sensitivity of gram staining to be 65%.

In a similar study by Bandaru Rao et al[18], Gram staining revealed the probable etiological agent in 80% cases.

Yield of bacteria on a gram stain depends on several factors such as number of organisms present, prior use of antibiotics, techniques used for smear preparation like centrifuged deposit,cytospin,direct smear etc, staining techniques and observer skill and experience.

In this study, Latex test revealed the organisms in 13 (61.9%) cases which correlates well with various other Indian studies[19]. H. influenza is the most common organism isolated in this study.Latex test can be useful to detect the organisms where the antibiotics are used early before CSF analysis.

CSF culture was positive in 3 (14.3%) cases. Several Indian studies report low CSF culture positivity ranging from 6 to 50%.

Various reasons for low yield of bacteria on culture are prior antibiotic therapy, delay in transport of specimens to the lab, non availability of special media for specific pathogens and lack of 24 hrs facility for processing CSF samples.

No case of meningococcal meningitis was reported during the study period .This may be due to low prevalence of meningococcal meningitis in children except during the epidemics.

Correlation of Gram stain & latex test with the CSF culture in identifying the organisms shows that Gram stain & latex are equally efficiacous in detecting the organisms and better than culture. ($p<0.05$).

This study shows that simple Gram stain combined with the latex test will give a rapid diagnosis on the etiology of bacterial meningitis.

Storring and synder[31] in their study of imaging in children with bacterial meningitis reported ventricular dilatation in 75%, subdural collection in 25%, cerebral edema in 9%, ischaemic infarcts in 19%.

In this study, most common finding observed was leptomeningeal enhancement 15 (71.4%) cases. One case of subdural hygroma[32] was found which is positive for H.influenza. Other findings were infarct in 1 case and normal study in 1 case.

We must anticipate complications while treating bacterial meningitis if there is lack of satisfactory response to antibiotics in the form of slow defervescence ,reappearance of fever while on treatment,persistence of seizures, reappearance or persistence of bulging fontanelle,deterioration of mental status or any increase in head circumference.

Neuroimaging studies are typically used to monitor the complications such as subdural effusion ,hydrocephalus ,infarction etc.Imaging studies in acute meningitis may provide normal findings. In this study , one case had a normal imaging.

Imaging studies does not prove or exclude the presence of acute meningitis.It is the CSF analysis which is the single most important diagnostic study.

TBM:

TBM occurs in 7-12% of tuberculous patients in developing countries.[33]

Wilson et al[34] in their series of cases of TBM, highest age group incidence observed was between 3-9 yrs which correlates with our study where the highest incidence of TBM were in the age group 3-8 yrs. The youngest child reported in this study was 9 months old.

Of these 21 cases,10 (47.6%) were males and 11 (52.4%) were females.

Gold standard for diagnosis of TBM is isolation of AFB bacilli in CSF which is usually rare. In this study the diagnosis of TBM was made by AIIMS criteria [16]

Contact H/o was present in 12 (57.1%) cases which correlates with Yaramis et al study[35].

In a study on the clinical profile of TB meningitis by Thilothammal et al[36] contact H/o was present in 40% cases.

Thilothammal et al [36] in their study observed that Mantoux positivity was present in 13% whereas in this study it was only 5%.

In a retrospective study of TBM in AIIMS[37], BCG scar was present in 50% which correlates with our study.

ICMR BCG trials[38,42] in chingelput also report that BCG offers no protection against primary TB infection or its progression to severe forms which correlates with our study (pvalue < 0.05).

Mathur et al[39] in a comparative study between BCG vaccinated and non vaccinated groups of patients could not find any significant difference in clinical pattern of TB which correlates well with this study.

In the study by Thilothammal et al [36]on TB meningitis, chest x ray was abnormal in 30% cases whereas in this study x ray was normal in all the cases.

Lymphadenopathy which is a common feature of primary TB infection in children cannot be detected by routine chest x ray.

CT is superior in detecting the lymphadenopathy[40] which may explain the observation of normal x ray findings in this study.

Elevated protein levels in CSF was observed in 16 (76.2%) cases which has a better diagnostic value in TBM than glucose values.

CSF sugar content may be normal(or) low in TBM and this alone should not be used as an evidence for (or) against TBM [34]. CSF protein values depends upon clinical stage of the disease. [36]

Bacteriological confirmation of diagnosis of TBM varies from 10% to 70% in various Indian studies .

In this study, AFB bacilli was not identified in any case which may be due to failure to examine a large volume of CSF and lack of fluorescent microscopy as a routine examination.

ADA level in CSF was done only in 3 cases. Two cases shows an increased value and one case shows normal value [41]. Cut off values of more than 8 IU/L favors the diagnosis.

In this study, hydrocephalus was the most common finding observed 42.9% in the neuro imagingwhich correlates with a study conducted at AIIMS (2000-2004).

In an recent analysis of TBM patients at AIIMS, CT abnormalities detected were hydrocephalus 65.2%,basal exudates 47.8%,cerebral edema 21.7%,infarcts 34.8%,tuberculoma 30.4%.

Ravenscroft et al [43] in their study showed that 16% of patients with TBM had associated intracranial granuloma. In our study one case of TB meningitis showed associated intracranial granuloma in MRI.

Imaging was normal in 9.5% of cases in this study which correlates with Ozates et al study. [44]

Imaging modalities have greatly enhanced the diagnostic accuracy of neurotuberculosis but not pathognomonic for the disease.

VIRAL ENCEPHALITIS:

In this study, 10 cases of viral encephalitis were observed. Out of the 10 cases, 5 cases of herpes viral encephalitis,3 cases of JE were observed.The highest incidence of cases were in the age group between 3 to 8 years.

Imaging studies remains the diagnostic modality of choice.Out of the 5 cases of Herpes encephalitis,CT shows normal study in 1, leptomeningeal enhancement in 1, cerebral edema in 2

and hypodense lesion in 1 case. None of the cases was picked up by CT. MRI detects the diagnostic lesions in the temporal lobe in all the cases.On comparison of CT and MRI ,herpes viral encephalitis was better diagnosed by MRI ($p < 0.05$).

In this study, 3 cases of Japanese encephalitis were observed. Serology was positive for JE in the CSF sample and in the blood in all the three cases. Imaging does not reveal the specific basal ganglia, thalami involvement in any case. Imaging abnormalities were cerebral edema (1) and leptomeningeal enhancement(2).

Seasonal outbreaks and epidemics of JE was not observed during the study period probably due to JE vaccination compaign undertaken in Madurai district 1 year back.

All the 3 cases of JE observed in this study had not undergone the vaccination.

CSF cell count was normal in 6 out of 10 cases of viral encephalitis in this study which could not be explained.

Out of these 6 cases with normal CSF cytology, diagnosis was made by imaging (MRI) which detected the lesions in the temporal lobe suggestive of Herpes Viral encephalitis in 4 cases.

The diagnosis of JE encephalitis was made in the remaining 2 cases based on CSF JE serology positivity.

In this study, 29 cases had normal CSF cytology with the clinical picture of AES (after excluding febrile seizures first episode,CP, neem oil encephalopathy).

ADEM an inflammatory demyelinating disease involving CNS white matter can present with fever and altered sensorium mimicking encephalitis. ADEM accounts for one third of all known case of encephalitis. [23,45]

In the case series on ADEM [23], 3 out of 7 cases presented with altered sensorium, and CSF abnormality were noted in 2 cases whereas in this study, out of 9 cases of ADEM, 5 cases presented with altered sensorium and CSF abnormality in the form of mild elevation of proteins and CSF lymphocytosis was observed in 3 cases .

In the case series on ADEM [23],youngest age group reported was about 8 months old whereas in this study the youngest age group in which ADEM was observed was 4 months old.

CT may be normal in ADEM where MRI is the diagnostic modality of choice which reveals the characteristic lesions in T2 weighted images. [24]

Haemorrhagic forms of ADEM have been reported [22]. In this study, among the 9 cases, one case had haemorrhagic type of ADEM in which CSF cytology showed the presence of RBC's and the diagnosis was made by imaging.

On the comparison of CT vs MRI in the diagnosis of ADEM, MRI remains the better imaging modality of choice.MRI should be considered early in patients with acute onset unexplained encephalopathy with or with out focal neurological deficit.

There are case reports of cerebral malaria presenting with symptoms of diffuse meningo encephalitis and meningeal signs.[27]

In this study, we had one case of cerebral malaria. 7 yr old female child from Paramakudi (malaria endemic area), was admitted with the clinical picture of AES. In this case, CSF analysis and imaging being normal simple peripheral smear examination revealed the diagnosis. Smear was positive for mixed infection of Pl.Vivax and Pl. falciparum.

In the clinical picture of AES, residing from an malaria endemic area, presence of anaemia with splenomegaly are important clues towards the diagnosis of cerebral malaria. This child was treated with artemesinin combination therapy (ACT), other supportive measures and a dramatic improvement was observed with in 36 hours.

All efforts must be made to diagnose cerebral malaria by examining peripheral smear repeatedly and also by using serological tests in cases where index of suspicion is high.

Dengue infection is one of the known cause of acute febrile encephalopathy in children.[21,46]

In this study, we report 2 cases of Dengue infection presenting as AES. Causes of altered sensorium in Dengue infection may be due to shock, hyponatremia, intracranial bleed, hepatic involvement , kidney failure and rarely encephalitis.

In this study, for both the cases CSF antibodies for dengue virus was negative which possibly excludes the encephalitis caused by dengue virus itself.

In this study, we had 2 cases of HIV presenting as AES. One case a known case of HIV on ART presented with AES. Imaging

reveals HIV vasculitis[49]. Other case had progressive encephalopathy imaging showing cerebral atrophy clinical suspicion clinches the diagnosis and confirmed by ELISA.

Vardhaman S. Udgirkar et al[50] in his article on the neurological manifestations of HIV infection reports that HIV encephalopathy can be a presenting manifestation among the infected children.

Brain abscess is an important differential diagnosis in children with unexplained fever, altered sensorium ,increased ICP and focal deficit.[47]

CSF examination may be normal or may show mild elevation of proteins and WBC'S.Imaging remains the diagnostic modality of choice[48].

In this study, one case of brain abscess was observed. 1 ½ years old male child admitted with fever, altered sensorium and there was no pupillary abnormalities, focal deficits or signs of increased ICP such as papilledema and so CSF analysis was done. CSF being normal, CT revealed the diagnosis and child was transferred out to neuro surgery department for the drainage of abscess.

Toxic encephalopathy is one of the cause for non infectious encephalopathy in children. Neem oil encephalopathy is one of the important cause in the southern region of Tamilnadu .

In this study, 4 cases of neem oil encephalopathy were observed.

Another case of toxic encephalopathy in this study includes a case of carbamazepine toxicity presented with altered sensorium and seizures and the child was treated symptomatically, regained consciousness with in 24 hours.

In this study, 5 cases of Acute encephalopathy for which etiological diagnosis could not be found due to non availability of serum ammonia, serum lactate etc.

In this study, we had 5 cases with post meningitic sequalae in the neuroimaging studies where the CSF analysis was normal.

In this study, we had 29 cases with the clinical picture of AES and CSF analysis being normal, but showing abnormalities in the neuroimaging studies 19 (65.5%) in CT and 22 (77.8%) in MRI.

This study shows clinical suspicion along with the appropriate neuroimaging studies picks up the etiological diagnosis in the group of patients presenting as AES with normal CSF analysis.

CONCLUSIONS

- CSF analysis helps in the differential diagnosis of AES to a great extent.
- The incidence of pyogenic meningitis is highest in < 1 yr of age group.
- The most common organism isolated is H.influenza.
- Gram stain & latex test are equally efficacious in identifying the organism.
- Protein levels in CSF has a significant diagnostic value than glucose levels.
- The role of neuroimaging in pyogenic meningitis is to detect the complications like subdural effusion,hydrocephalus etc.
- TBM is common in the age group between 3 to 8 yrs.
- Similar to pyogenic meningitis, protein values has a significant diagnostic value than glucose levels.
- No significant difference was observed between those with BCG scar & those without BCG scar.

- Contact history was positive in only 57% of cases with TBM.
- Mantoux positivity was observed in only 4%.
- AFB bacilli was not identified in any case.
- Hydrocephalus was the most common finding observed in neuroimaging studies in TBM.
- MRI is the better imaging modality of choice for the diagnosis of herpes viral encephalitis.
- JE positivity was observed in 30% of viral encephalitis.
- When CSF analysis is normal, appropriate neuro imaging helps in the differential diagnosis of AES.

LIMITATIONS

- Microbiological confirmation of pyogenic meningitis was not possible in all cases due to prior use of antibiotics, technical difficulties in our set up.
- Imaging could not be done in all cases due to poor general condition, short duration of stay, patient on ventilator support.
- Biochemical markers for TBM such as tuberculostearic acid (TSA) assay, bromide partition tests, LDH levels were not done.
- CSF DNA PCR analysis for Herpes Viral encephalitis were not done.
- Etiology for acute encephalopathy could not be found in some cases due to limited investigational facilities.
- Certain metabolic conditions (IEM, Reye's syndrome) could not be ruled out due to non availability of serum ammonia, lactate levels , ABG analysis.
- EEG could not be done in all cases.
- Follow up was not done to predict the long term outcome.

SUGGESTIONS /RECOMMENDATIONS

- All the investigations for complete metabolic work up should be made available in tertiary care hospitals.
- Electrophysiological studies such as EEG should be made available bed side for better diagnosis.
- Technical problems in the transport & processing of CSF samples needs further improvement for a better microbiological results.
- CSF cell count analysis should be done as a bed side investigation.
- Every clinician should be empowered with this skill as it's an extended part of clinical examination.

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Name : Age : Sex :

Address : Occupation: Income :

Presenting Complaints:

H/O Fever
H/O Convulsions
H/O Altered Sensorium
H/O Head Ache
H/O Vomiting
H/O Cough/Cold
H/O Diarrhoea
H/O Ear Discharge
H/O Rashes
H/O Toxin Ingestion
H/O Drug Intake
H/O Trauma
H/O Recent Vaccination
H/O Abnormal Odour In Breath/ Urine
H/O Contact With Tb

General Examination:

Vitals	Anthropometry
HR	Length/Height
RR	Wt
BP	HC
CRT	CC
Temp	MAC

O/E Child

Febrile
Hydration
Pallor
Icterus
Cyanosis
Clubbing
Generalised Lymphadenopathy
Pedal Edema
Anterior Fontanelle
Bcg Scar
Neurocutaneous Markers

Examination of Ear : Discharge, Perforation

Foci of Sepsis

Examination of CNS :

Higher Functions

Examination of Cranial Nerves

Examination of Motor System

	R	L
Bulk		
Tone		
UL		
LL		
Power		
UL		
LL		
DTR		

Superficial Reflexes

Sensory System

Cerebellar Signs

Meningeal Signs

Involuntary Movements

Fundus

Cvs:

Rs:

Abdomen:

Investigations:

Hb%	Urine Analysis
Tc	Blood Sugar
Dc	Urea
ESR	Creatinine
BT	Serum Electrolytes
CT	Liver Function Test
Peripheral Smear	
Blood Culture	Gastric Juice for AFB
CRP	Serology for JE

Mantoux	Chest X Ray

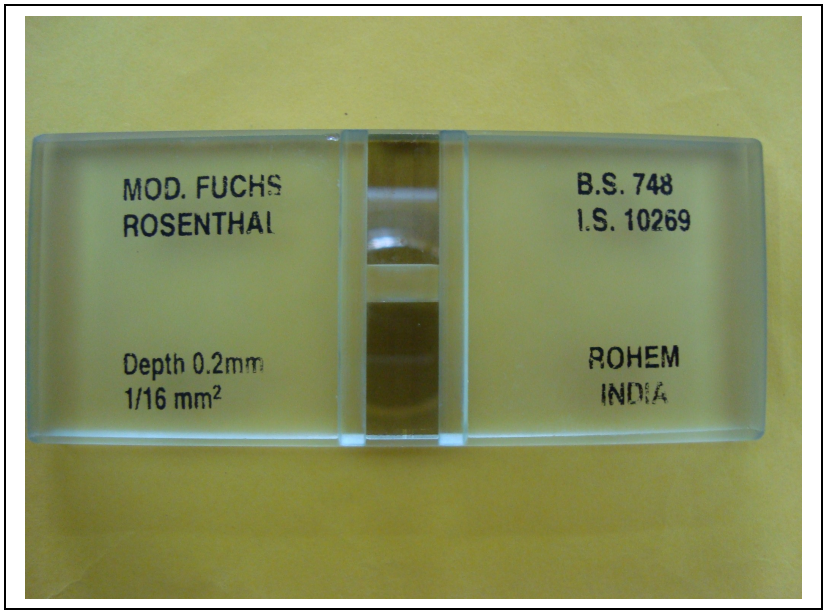
CSF Analysis :

Cell Count	Biohemical Analysis
Gram Stain	
AFB Stain	
Culture	
ADA Assay	
Cobweb Coagulum	

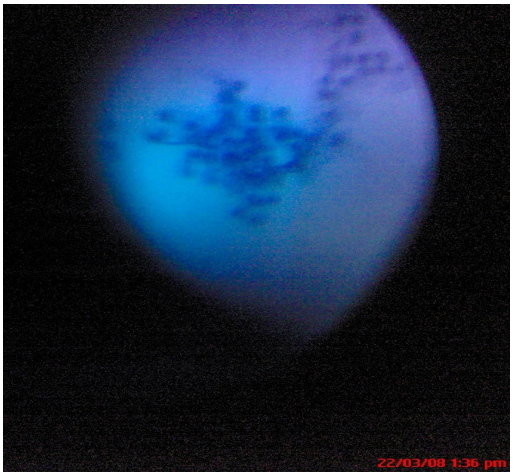
Imaging Studies:	CT/MRI
Special Tests:	Serum Ammonia, Lactate, ABG

ABBREVIATIONS

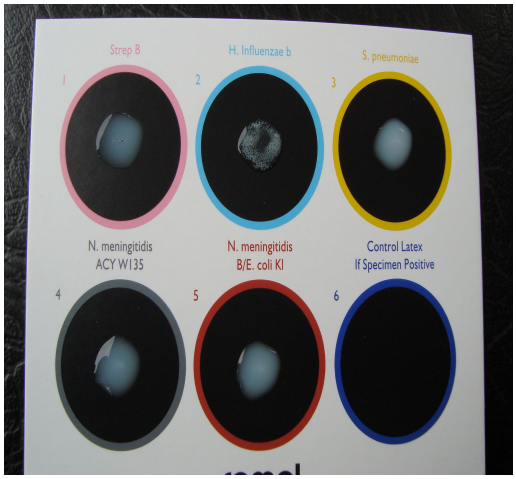
ABG	Arterial Blood Gas Analysis
ADA	Adenosine Deaminase Assay
ADEM	Acute Disseminated Encephalomyelitis
AES	Acute Encephalitis Syndrome
AFB	Acid Fast Bacilli
BCG	Bacillus Calmetie Guerien
CMV	Cytomegalo Virus
CNS	Central Nervous System
CP	Cerebral Palsy
CRP	C Reactive Protein
CT	Computerised Tomography
CVT	Cerebral Venous Thrombosis
EEG	Electro Encephalography
HIV	Human Immuno Deficiency Virus
HSV	Herpes Simplex Virus
ICP	Intra Cranial Pressure
IEM	Inborn Error Of Metabolism
IL	Inter leukins
JE	Japanese Encephalitis
LAT	Latex Agglutination Test
LDH	Lactate Dehydrogenase
LP	Lumbar Puncture
MRI	Magnetic Resonance Imaging
MSUD	Maple Syrup Urine Disease
PCR	Polymerase Chain Reaction
PLEDS	Paroxsymal Lateralised Epileptiform Discharge
RBC	Red Blood Cell
TBM	Tuberculous Meningitis
TNF	Tumour Necrosis Factor
WBC	White Blood Cell



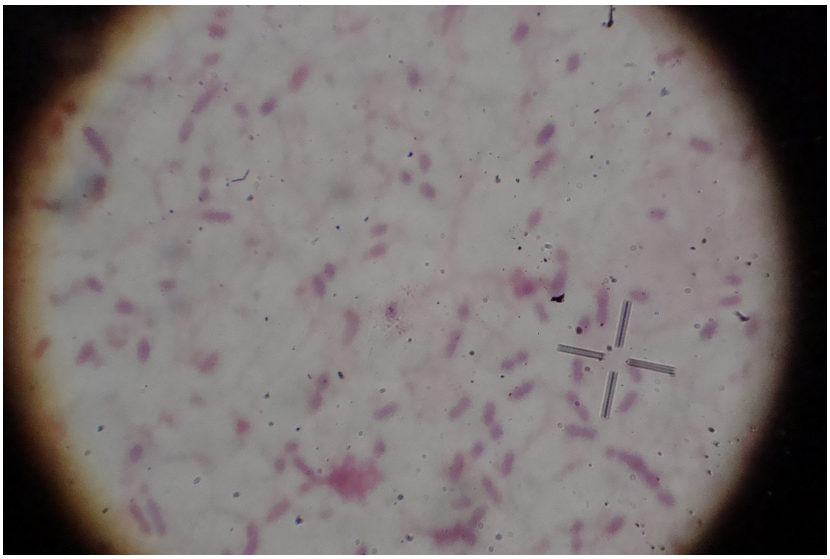
CSF POLYMORPHOLEUCOCYTOSIS



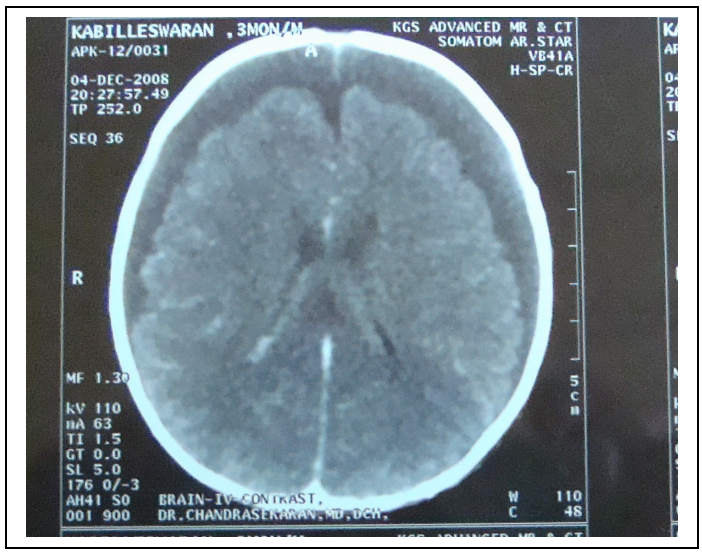
LATEX TEST



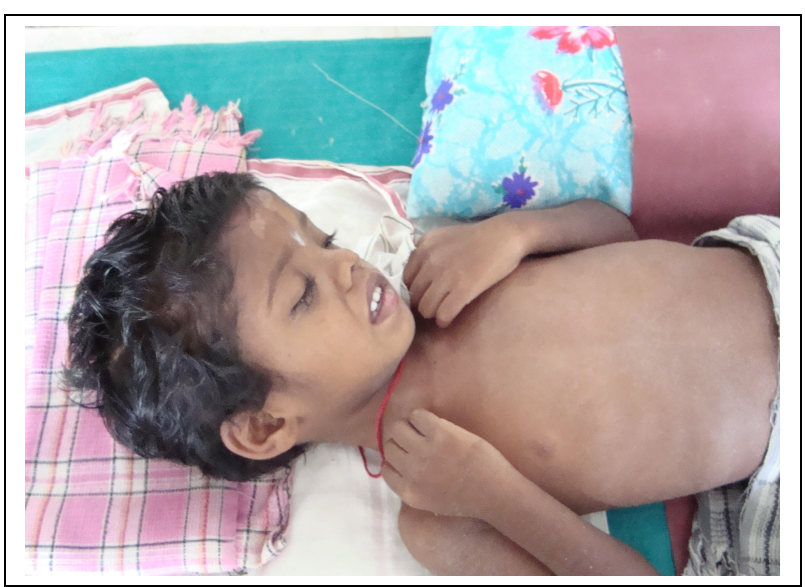
GRAM STAIN H. INFLUENZA



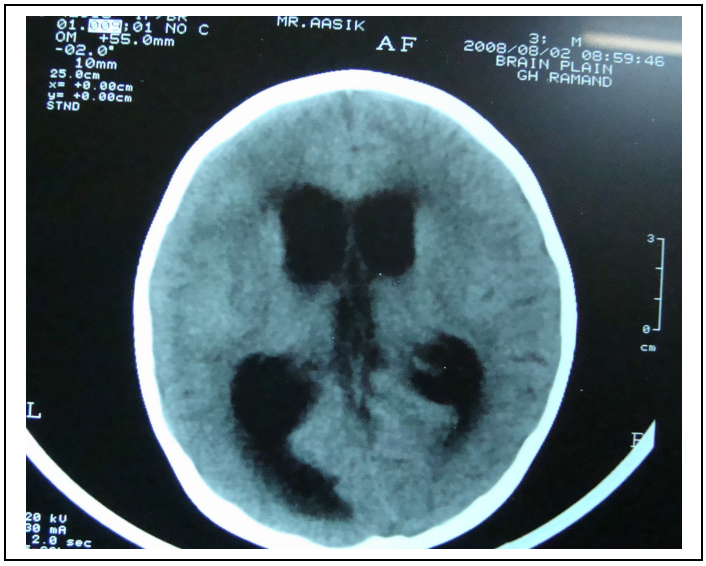
SUBDURAL EFFUSION



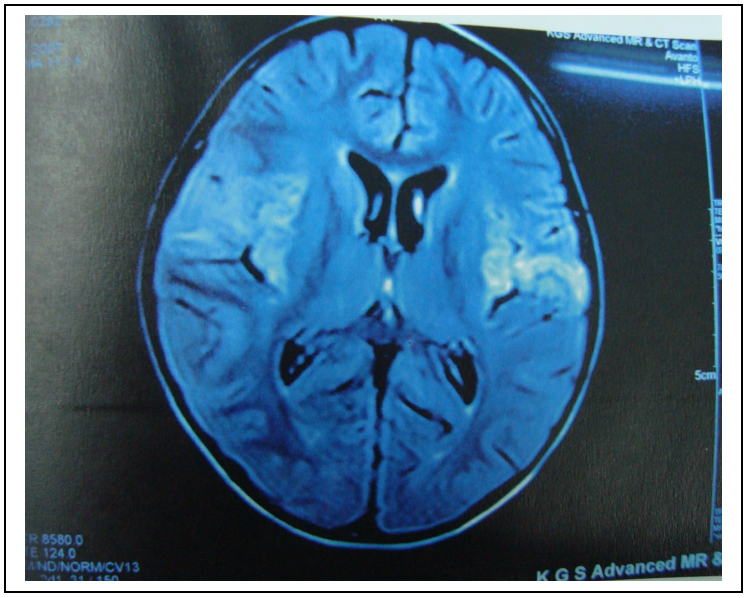
CHILD WITH TB MENINGITIS



HYDROCEPHALUS



MRI – HERPES ENCEPHALITIS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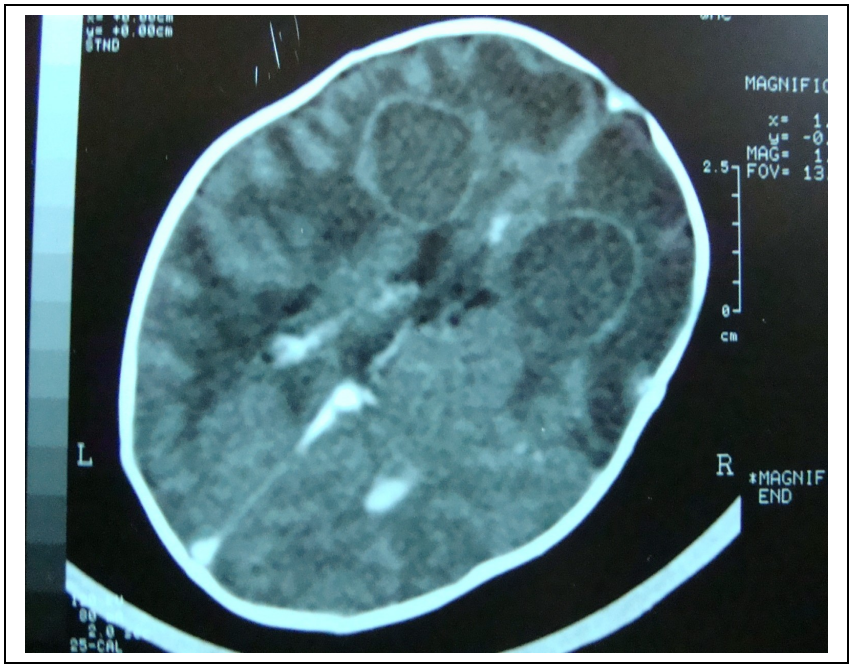
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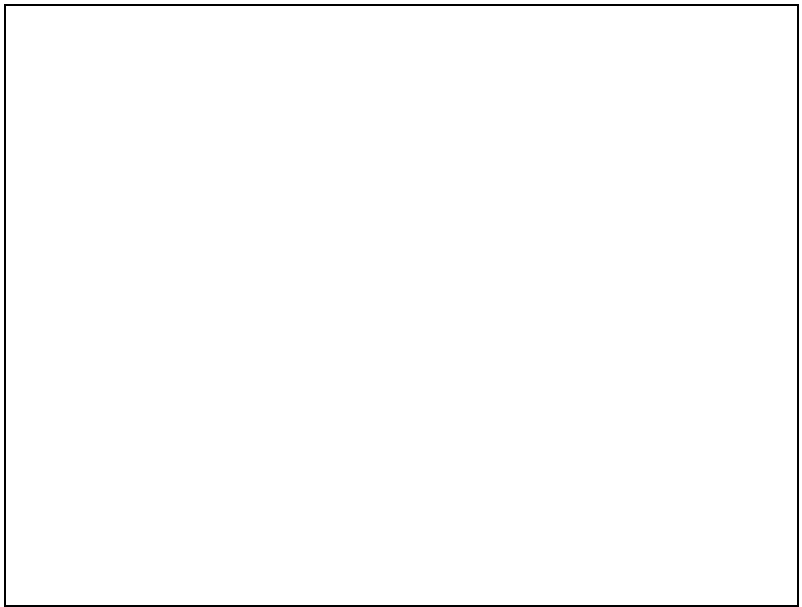

ADEM

HIV - CEREBRAL ATROPHY