CLINICAL EPDIMOLOGICAL PROFILE OUTCOME OF ACUTE MENINGO ENCEPHALITIS CHILDREN AGE 1 MONTH TO 12 YEARS

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CERTIF ICATE

CERTIFICATE

Certified that this dissertation entitled **"CLINICAL EPDIMOLOGICAL PROFILE OUTCOME OF ACUTE MENINGO ENCEPHALITIS CHILDREN AGE 1 MONTH TO 12 YEARS"** is a bonafide work done by Dr.N.SASIKUMAR, M.D., Post graduate student of Pediatric Medicine, Coimbatore Medical College & Hospital, Coimbatore – 641018 during the academic year May 2016 – 2018.

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Certified that this dissertation entitled **"CLINICAL EPDIMOLOGICAL PROFILE OUTCOME OF ACUTE MENINGO ENCEPHALITIS CHILDREN AGE 1 MONTH TO 12 YEARS"** has been conducted by me at Department of pediatrics, Coimbatore Medical College and Hospital, under the guidance and supervisor of my Guide **Prof.Dr.A.Lakshmanaswamy, M.D., D.C.H.,** It submitted in part of fulfillment of the award of the degree of M.D Pediatrics for the MAY 2018 examination to the held under the Tamil Nadu Dr.M.G.R.Medical University, Chennai, This has not been submitted previously by me for the award of any degree or diploma from any other university.

(Dr.N.SASIKUMAR)

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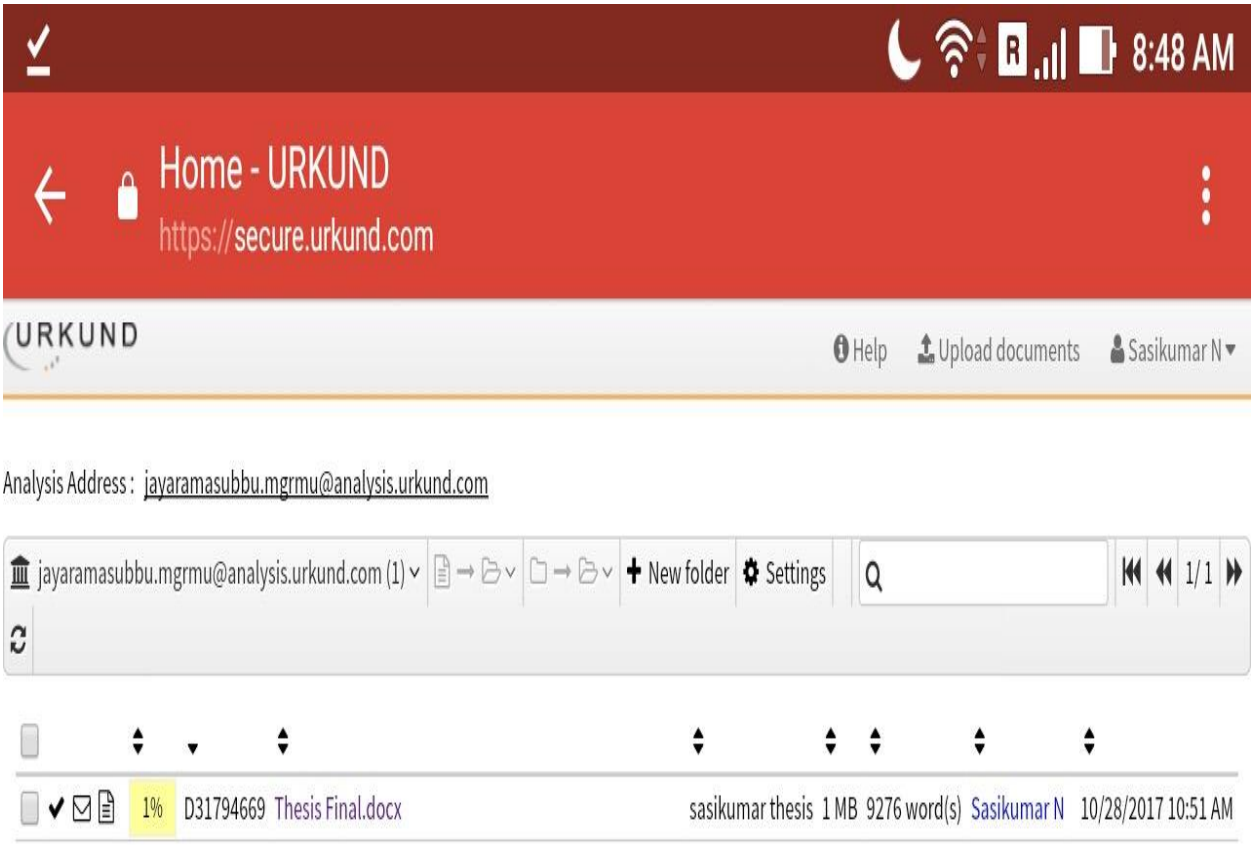
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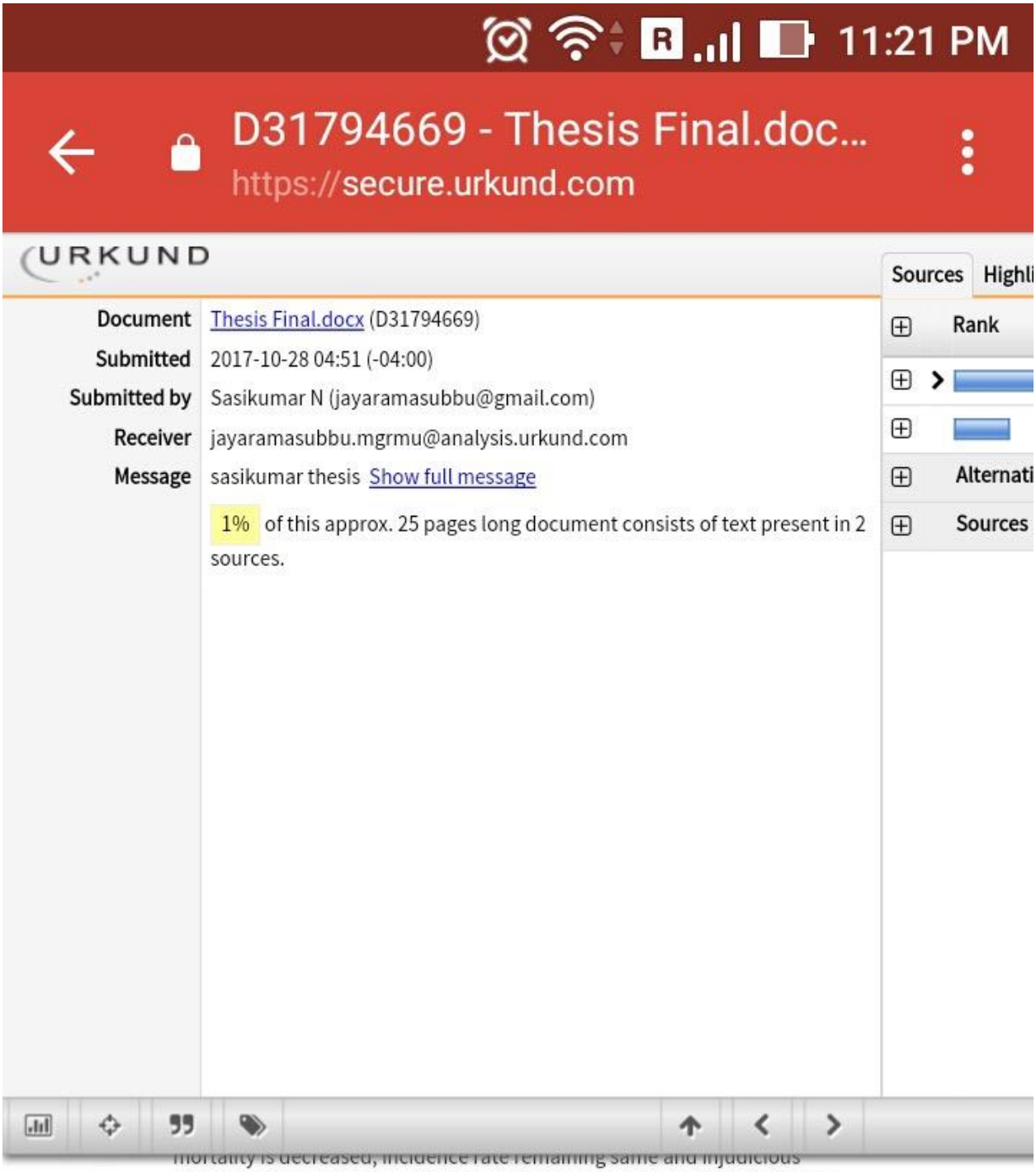

mortality is decreased, incidence rate remaining same and injudicious use of antibiotics at suboptimal dose and in appropriate antibiotics is still major causes of death with mortality rate above 30%.

AIM

The Aim of my dissertation is to evaluate the clinico epidemiological profile and outcome of Acute meningoencephalitis in children age one month to 12 yrs.

OBJECTIVES

1. To assess the influence of epidemiological status of children and out come of the meningo encephalitis in children are one month to 12 yrs.

2. To find out the association between the CSF analysis and out come of meningo encephalitis.

REVIEW OF LITERATURE

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ABBREVIATION

FND	-	Focal Neurological Deficit
HSV	-	Herpes Simplex Virus
JE	-	Japanese Encephalitis
CSF	-	Cerebro Spinal Fluid
CNS	-	Central Nervous System
TB	-	Tuberculosis
PMN	-	Poly Morpho Netrophils
Hib	-	Haemophilus Infection
HIV	-	Human Immuno Deficiency Virus
BBB	-	Blood Brain Barrier
SAS	-	Sub arachnoids Spare
TNF	-	Tumor Necrosis Factor
MMP	-	Matrix Mettallo Proteins
ICP	-	Intra Cranial Presser
CT	-	Computer Tomography
LP	-	Lumbar Puncture
MRF	-	Magnetic Resonance Imaging
PICU	-	Pediatrics Intents care Unit
WNU	-	West Nile Virus
EEG	-	Electro Enchaphalo Gram

INTORDUCTION

INTRODUCTION

Meningoencephalitis is an inflammatory process involving both the brain parenchyma and meninges. Meningitis indicates 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 and parenchymal involvement.

Meningoencephalitis primarily affects infants & children. Leading to increase in morbidity and adverse outcome like neurological damage, hearing impairments and poor school performance. It's one of the most frequent infections in infants and childhood. More then 2/3 of cases of meningitis in less than2years are due to poor immunity and high vascularity of brain.

Acute meningoencephalitisis characterized by acute onset of fever, change in mental status such as confusion, disorientation (or) inability to talk , coma, head ache, vomiting, FND(Focal Neurological deficit), photophobia, neck pain and new onset of seizures. Most common causative bacterial agents are Streptococcal pneumonia, H influenza, Nisseriameningitidis and most important viral agents are HSV (Herpes

simplex Virus), JE (Japanese encephalitis),Enterovirus ,Dengue ,Varicella and HIV.

Meningoencephalitis is a diagnosis to be made from clinical symptoms and signs along with CSF Analysis.

Mortality rate has come down and has decreasedto 40%. Even though mortality has decreased, incidence rate remains the same and injudicious use of antibiotics at suboptimal dose and use of inappropriate antibiotics is still a major cause of death.

AIM AND OBJECTIVES

AIM

The Aim of my dissertation is to evaluate the Clinico-epidemiological profile and Outcome of Acute meningoencephalitis in children agedone month to 12 yrs.

OBJECTIVES

1. To assessthe epidemiological profile and outcome of the meningo encephalitis in children from one month to 12 yrs.

2. To find out the association between the CSF analysis and outcomeofmeningo encephalitis.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Infection of the CNS Central Nervous System is the most common cause of mortality and morbidity in the developing countries due to poor immunization and poor socio economic status.Overcrowding and poor education in India is the most common cause for meningoencephalitis.Most common pathogen is virus, followed by bacteria and fungal infection. Severity of the infection depends upon the agent, age of the children, immunization status and epidemiological factors.

Acute CNS infection is diagnosed by clinical signs and symptoms like fever, altered sensorium, seizures, neck pain, photophobia and abnormal CSF finding.

Early suspicion and diagnosis is very important to reduce the morbidity and mortality of acute CNS infection.

TABLE

Conditions	Pressure	Leucocytes	Protein	Glucose
Normal	50 – 80 mm Hg	<5 Cells 75% Lymphocytes	20-45	more than 50% (or) 2/3 of serum glucose
Bacterial Infection	Increased (100-300)	100-1000 Cells neutrophil predominance (300 – 2000)	100-500	<40
Partially treated Bacterial Meningitis	Normal (or) Increased	5 – 10,000 cells. PMN + Mono Nuclear cells Predominance	100-500	Normal
Viral Meningitis	Normal (or) Increased (80-150)	Rarely morethan 1000 cells .PMN- early. Monocytes Later	50-200	Normal or<40
TB Meningitis	Increased	10-500 Cells Lymphocytes Predominance	100-300 ,increased when block is present	<50
Fungal Meningitis	Increased	5-500 Cells PMN Early Monocytes later	25-500	<50

PMN –Polymorpho Nuclear Cells

INFECTIONS OF THE CENTRAL NERVOUS SYSTEM

Acute Bacterial Meningitis

Etiology

The three most commoncausative organisms in children are Hemophilusinfluenza,NeisseiraMeningitidisand Streptococcal Pneumonia. Inneonates, the most common organisms are Gram negative bacilli, group B Streptococci and Listeria Monocytogens.

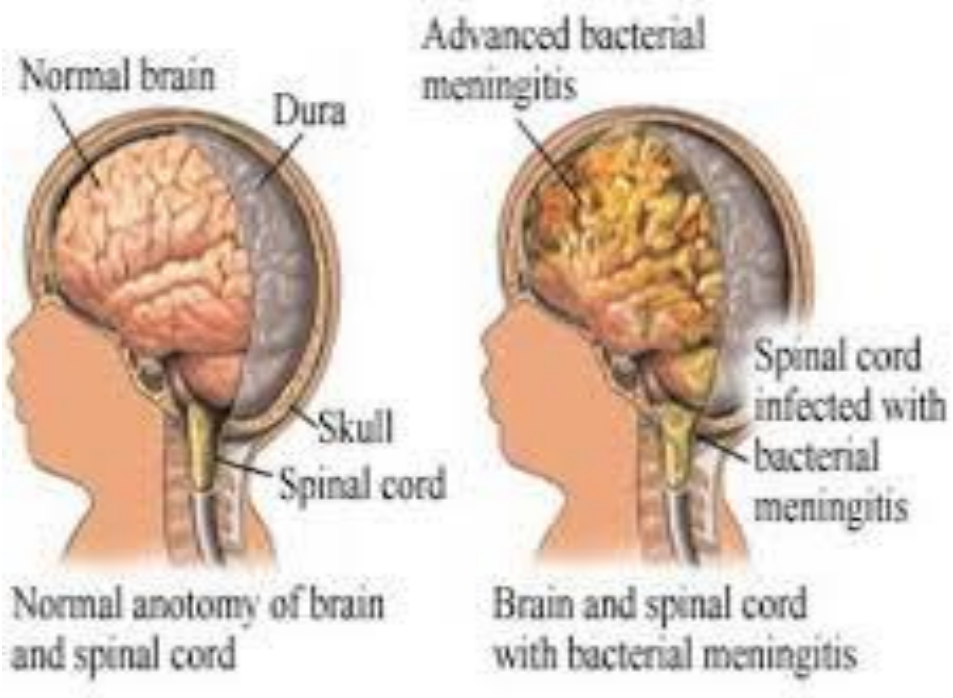
Bacterial meningitis may be sporadic or epidemic.It commonly occurs in infants and children although it can occur at any age. In developed countries a substantial reduction of H. Influenza and Pneumococcal meningitis has occurred, because of widespread immunization. However these organisms continue to cause meningitis in many developing countries. Poor socioeconomic conditions, overcrowding, malnutrition, and HIV further, contributes to the high incidence of meningitis in developing countries.

Hib meningitis occurs usually in children between 3 months to 3 years of age .Pneumococcal meningitis occurs throughout childhood and is particularly seen in children with Pneumonia, otitis media, sinusitis, CSF leaks, head injury, sickle cell disease and thalassemia major. Meningococcal meningitis occurs mainly in school age children and young adults. Meningitis caused by Hib, Pneumococcal and

meningococcal is rare in first 3 months of the because of trans placental transfer of protective maternal antibodies.

Pathogenesis

Bacterial meningitis occurs through the haematogenous route in most cases. Inhaled bacteria adhere to the nasopharyngeal mucosa, and evade mucosal host defense mechanisms and enter the blood stream, and CSF by penetrating the blood brain barrier (BBB). This is facilitated by various neurotropic and virulence factors. CNS infection can also occur from contiguous sites of infection such as otitis media, sinusitis and mastoiditis or through direct communication of the subarachnoid space (SAS) with the skin or mucosa as in head trauma, dermal sinuses and through infected CSF shunts.

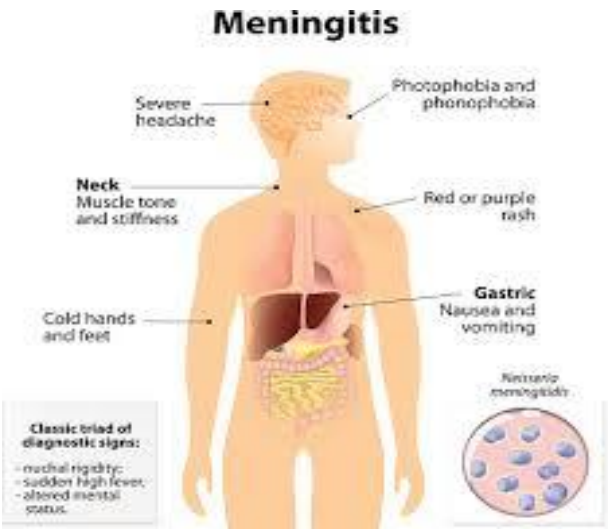


Normal anotomy of brain and spinal cord

Brain and spinal cord with bacterial meningitis

Bacterial multiplication and autolysis in the subarachnoidspace leads to the release of bacterial components that lead to a strong inflammatory response .Several inflammatory cytokines the chemokine's including interleukins and tumor necrosis factor (TNF) are produced that in turn lead to the release of other inflammatory mediators, including platelet activating factor, matrix metalloproteinase, nitric oxide and free oxygen radicals that lead disruption of the Blood brain barrier. Leukocytes pass from the blood into the CSF leading to CSF pleocytosis, and further release of various toxic mediators that adversely affect the cerebral blood flow and cerebral metabolism, and contribute to the development of cerebral edema.

The inflammatory exudates cover the brain cerebral blood vessels, causes vasculitis and thrombosis, ischemia may lead to cerebral infarction. Cerebral edema may be marked and may lead to cerebral herniation, neuronal damage finally occurs.

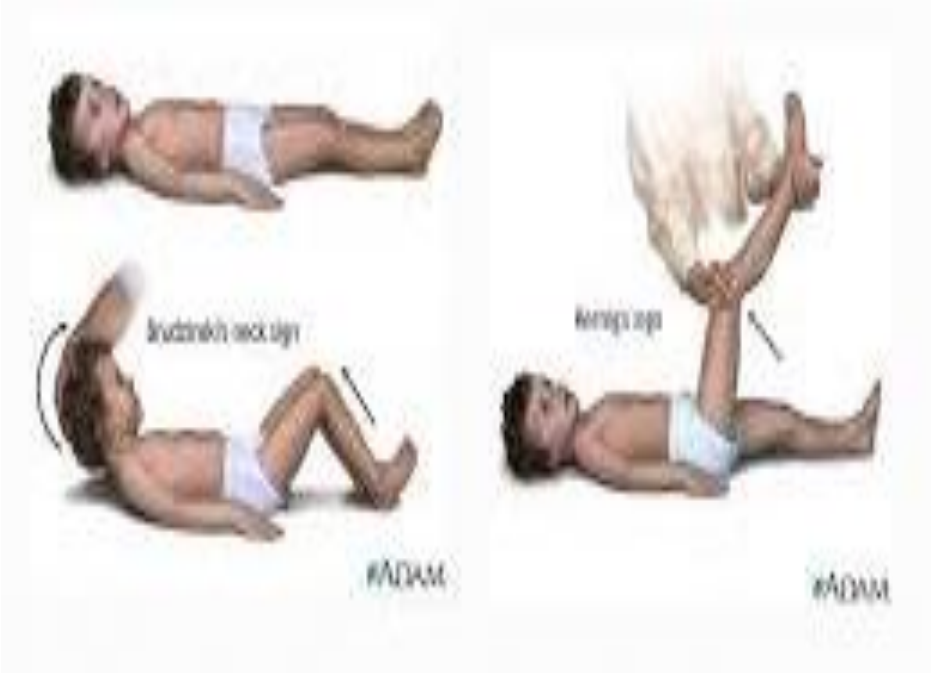


Typically meningitis presents acutely with fever, headache, vomiting altered sensorium, stiff neck, seizures and photophobia. The child may have a preceding upper respiratory infection, pneumonia or otitis media. Fever may be absent in very small infants in severely malnourished or immune compromised children and in children on previous antibiotic therapy. Younger children may not be able to complain of headache but may be irritable. Seizures may be the presenting feature in almost a third of children with meningitis but it may be recurrent and prolonged. Alteration of sensorium at earlier presentation may be minimal but worsens gradually with progression of the disease. In some cases of meningococcal meningitis it may be fulminant course with sepsis, shock (Water house Frederickson Syndrome) rapid progression, cerebral edema and raised intracranial pressure (ICP) evolving over a few hours may occur.

On examination, neck rigidity and other signs of meningeal irritation are seen. These include

- Kernig's 'sign; with thigh flexed on abdomen passive extension of knee produces pain in the back.
- Brudzinski's sign: Passive flexion of neck produces flexion of both lower limbs.

- Tripod Sign: In the sitting position the child supports himself with both arms extended behind the back which is kept straight.
- Knee – Kiss sign: The child cannot bend forward to kiss his knee.



Meningeal signs may be minimal or absent in neonates and young infants and in malnourished and deeply comatose children. In infants, a bulging fontanel is an important sign of meningitis. Symptoms and signs of raised ICP (headache, vomiting and respiratory abnormalities) may appear within 24-48 hours. Papilledema at initial presentation is uncommon and if present a CT scan must be done to exclude a mass lesion or a complication. Focal signs may be due to subdural collection, cortical infarction or cerebritis.

Assosciated findings such as a maculopapular or petechial rash in meningococcal infections .Otitis media or pneumonia in pneumococcal infections, and pustular skin lesions in staphylococcal infections may be seen.

As early diagnosis of meningitis is extremely important. It should be suspected in any child with lethargy, refusal of feed, stiff neck and seizures. It is important to note that absence of fever or meningeal signs does not exclude the diagnosis of bacterial meningitis.

Differential Diagnosis

a) Acute onset fever and Meningism

Aseptic meningitis ,pneumonia with right upper lobe involvement ,Retropharyngeal abscess and Cervical lymphadenitis

b) Encephalitis

Reye's Syndrome, Metabolic problems, hepatic encephalopathy, intoxications and other causes of non traumatic Coma. Cerebral malaria is an important differential diagnosis in endemic regions. Blood films for malarial parasites should be taken in children with spleenomegaly and anemia.

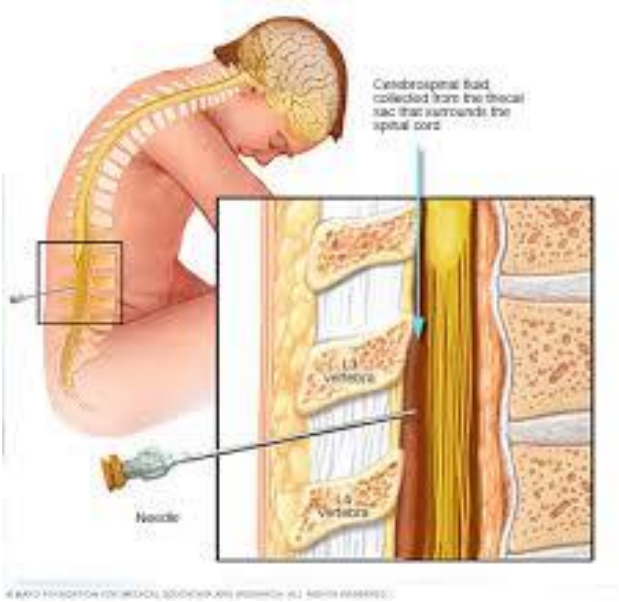
c) Focal Signs

Cerebral abscess ,herpes encephalitis, intracranial bleeds and other space occupying lesions need urgent neuroimaging because of subacute presentation. Tubercular, fungal and parasitic meningitis.

Cerebral fluid examination

CSF examination is essential for the diagnosis of meningitis. Lumbar puncture (LP) should be done as early as possible in all cases of suspected meningitis and also in the following.

- Neonates and very young infants who present as nonspecific sepsis.
- Children with febrile seizures upto 18 months of age or if the child looks ill or is on previous antibiotics.



Contraindication of LP include

- Raised ICP
- Focal Neurological symptoms or signs
- Shock / Cardio respiratory instability
- Thrombocytopenia (platelet count < 40,000mm^3)
- Coagulation disorder
- Local infection at LP Site.

Appropriate antibiotics should be given early in all cases of suspected meningitis even if the LP is delayed. Early antibiotic administration for 24 hours does not significantly alter the CSF cellular response but may decrease the yield on culture and Gram stain.

CT scan before the LP is not routinely needed. It is indicated in children with focal neurological symptoms , signs ofpapilledema,critically raised ICP or suspicion of a mass lesion. CT is normal in most cases of bacterial meningitis, including those with subsequent herniation, a normal CT does not rule out raised ICP.

Typically the CSF pressure is raised there is polymorphonuclear leukocytosis, decreased glucose and increased protein concentration. The cell count may vary from a few to hundreds to over a thousand leukocytes. Almost 90% cases have a CSF cell count over 100/mm^3.

In developing countries the reported CSF cell counts are not very high in most cases. A cloudy CSF under pressure is almost diagnostic of bacterial meningitis. Cell count should be done within half an hour of CSF collection .In normal children the CSF has 0-5 mono nuclear cells /mm^3, poly morph nuclear cells are very rarely seen. Presence of even a single polymorphonuclear cell in the clinical setting of meningitis should be considered significant.

The CSF glucose levels are less than 40mg/dl in more than half the cases, a CSF to serum glucose ratio of 0.4 or less is 80% sensitive and 98% specific in children's above 2 months of age. The blood glucose should be collected before the LP.

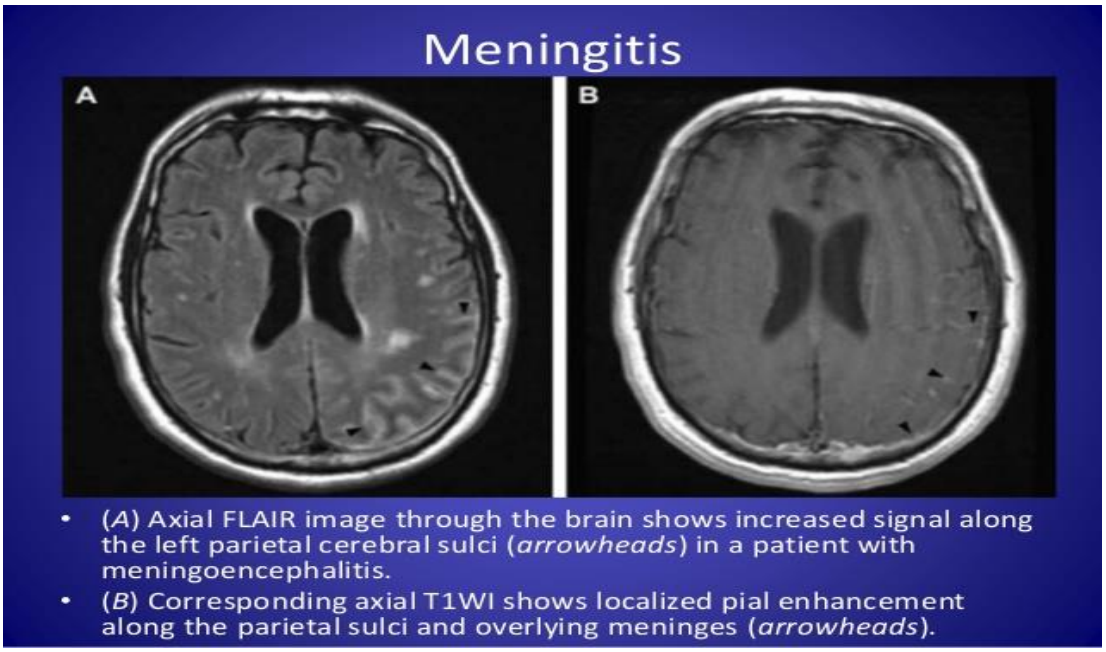
At times the CSF can be completely normal in early stages of bacterial meningitis particularly in neonatal meningitis a repeat LP after few hours may show abnormalities. Antibiotic therapy must therefore be started in all cases with a strong suspicion of bacterial meningitis.

Occasionally an initial poly morph nuclear response can be seen in viral meningitis and an initial lymphocytic response in bacterial meningitis. The CSF sugar is normal in most of viral meningitis.

Course and complications

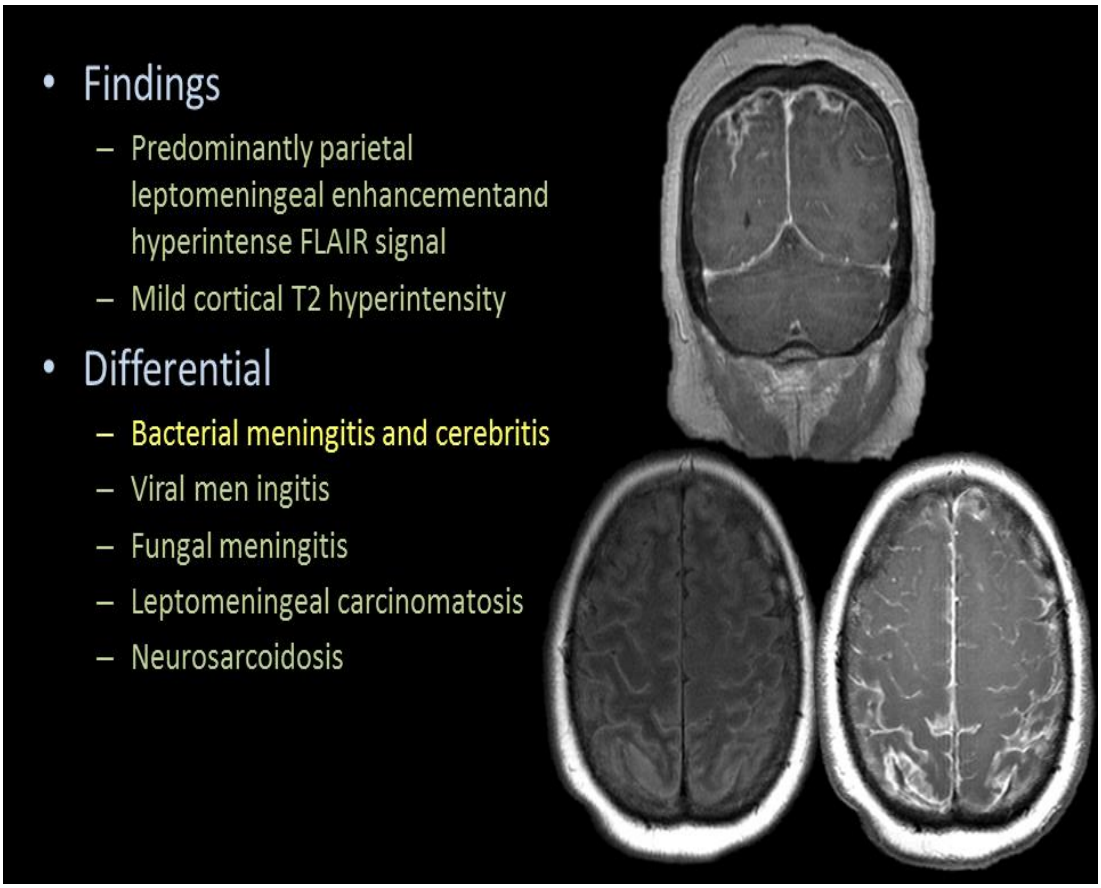
If treated properly children improve within 48-72 hours and the fever comes down within 4-5 days. Persistent fever (> 10 days) may be due to thrombophlebitis, subdural effusion,spread of infection. (Such as Pneumonia, arthritis or osteomyelitis) drug fever and rarely resistant organisms.

CT Scan during the therapy is indicated in a child who does not show clinical improvement ,has sudden unexplained deterioration, new onset seizures or focal neuralgic signs, signs of raised ICP, persistent fever or enlarging head subdural effusions, infarct, hydrocephalus, celebrities, or cerebral abscess may be detected. MRI detects parenchymal complications earlier than CT. Ultrasound of the head is helpful in neonates and in infants with open fontanel.



Complications of Meningitis

- Late onset seizures
- Subdural empyema
- Infarcts, cerebritis and brain abscess
- Hydrocephalus
- Cranial nerve involvement
- Sensorineural deafness
- Diabetisinsipidus
- Spread of infections to distant sites (pneumonia, pericarditis, arthritis and osteomyelitis)



Management

Appropriate IV antibiotics and supportive therapy should be started as soon as possible. Close monitoring is essential to detect and manage any acute life threatening complications such as shock or raised ICP. Ideally this is done in a pediatric intensive care unit (PICU) until the child is stable.

This should be broad enough to cover all the likely pathogens according to the age of the child and prevalent epidemiology and resistance patterns and its susceptibility .Antibiotic therapy is targeted accordingly.

The standard recommendation for the duration of antibiotics is 10-14 days for Pneumonia and H. influenza and 7 days for N. Meningitides. Shorter duration of antibiotic therapy (5 days) have also been found effective. A minimum of 3 weeks of antibiotics are required for Gram negative bacilli, staphylococcal meningitis and for neonatal meningitis.

With proper treatment the CSF culture and Gramstain become negative within 24-48 hours and the CSF glucose normalized over 72 hours. The increase in cells and proteins may persist for several days. A repeat LP either during treatment or at the end of therapy is not routinely needed unless there is appearance of new symptoms or signs.

Supportive Treatments

Airway, breathing and circulation must be maintained. Shock, raised ICP, seizures and status epileptics must be urgently and appropriately managed. Enough fluids should be given to maintain normal blood pressure and adequate cerebral perfusion. Electrolyte imbalances should be corrected.

Corticosteroid Therapy

Early dexamethasone therapy (0.15mg/kg/6hours x 4 days) has been recommended in developed countries as it reduces mortality and severe hearing loss and neurological sequelae in case of Hib and penumococcal meningitis. However beneficial effect of corticosteroids has been found in children.

Oral Glycerol

Oral glycerol for the first 48 hours (6kg/1day/ q 6 hour) may reduce neurological sequelae.

The role of intravenous immunoglobulin

The role is limited to children with immune deficient states and in some cases of neonatal meningitis.

Prognosis

Coma, raised ICP, status epilepticus, shock, and respiratory depression are important predictors of mortality and morbidity. Neurologic sequelae including hearing loss,hydrocephalus, spasticity ,visual and cognitive deficits and developmental delay are common.

Prevention

To prevent secondary meningitis, children with Hib or meningococcal meningitis should be isolated for 24 hours.

Chemoprophylaxis

Hibmeningitis : Rifampicin (20mg / kg / day) twice daily for 4 days) for all household contacts it there is at least one unvaccinated contact less than 4 years old. Rifampicin for the index case before discharge if ampicillin and / or chloramphenicol were used as they do not eradicate H Influenzae.It is not needed if ceftriaxone was used. In Meningococcal disease, Rifampicin (10mg / kg / 12hours for 2 days) for household and day care contacts .Ceftriaxone single im dose (125-mg for children < 12 years and 250 mg for older children and adults). Ciprofloxacin 500mg or azithromycin 500mg single dose in adults.

Primary prevention : It is possible by immunization against the common pathogens causing meningitis.

VIRAL MENINGO ENCEPHALITIS

Viral Meningoencephalitis is an acute inflammatory process involving the meninges and to a variable degree, brain tissue.

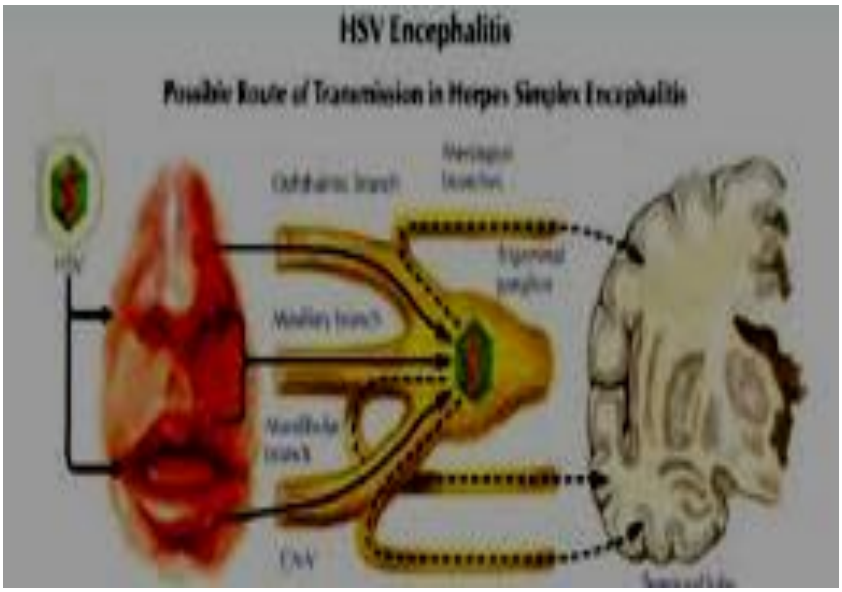
Etiology

Enteroviruses are the most common cause of viral meningo encephalitis. The severity of infection caused by enteroviruses ranges from mild, self – limited illness with primarily meningeal involvement to severe encephalitis resulting in death or significant sequelae. Parechoviruses may be an important cause of aseptic meningitis or encephalitis in infants. The clinical manifestations are similar to that of the enteroviruses with the exception of more severe MRI lesions of the cerebral cortex andat times an absence of a CSF pleocytosis.

Arboviruses are arthropod – borne agents, responsible for some cases of meningoencephalitis during summer months. Mosquitoes and ticks are the most common vectors, spreading diseases to humans.

Several members of the herpes family of viruses can cause meningo encephalitis. Herpes simplex virus (HSV) type is an important cause of severe sporadic encephalitis in children and adults. Brain involvement usually is focal . Severe encephalitis with diffuse brain

involvement is caused by HSV type 2 in neonates who usually contract the virus from their mothers at delivery. A mild transient form of meningoencephalitis may accompany genital herpes infection in sexually active adolescent, most of these infections are caused by HSV type 2, varicella –zoster virus may cause CNS infection in close temporal relationship with chickenpox. The most common manifestation of CNS involvement is cerebellar ataxia and the most severe is acute encephalitis. After primary infection varicella – zoster virus becomes latent in spinal and cranial nerve roots and ganglia, expressing itself later as herpes zoster, sometimes with accompanying mild meningoencephalitis. Cytomegalovirus infection of the CNS may be part of congenital infection or disseminated disease in immune compromised hosts. Human herpes virus 6 can cause encephalitis, especially among immune compromised hosts.



Mumps is a common pathogen in regions where mumps vaccine is not widely used. Mumps meningoencephalitis is mild but deafness from damage of the 8th cranial nerve may be a sequelae. Meningoencephalitis is caused occasionally by respiratory viruses (adenovirus, influenza virus, parainfluenzavirus) rubeola, rubella, or rabies. It may follow live virus vaccinations against polio measles, mumps, or rubella.

Pathogenesis and Pathology

Neurological damage is caused by direct invasion and destruction of neural tissues by actively multiplying viruses or by a host reaction to viral antigens. Tissue sections of the brain generally are characterized by meningeal congestion and mononuclear infiltration ,perivascular cuffs or lymphocytes and plasma cells, some perivascular tissue necrosis with myelin breakdown, and neuronal disruption in various stages including ultimately neuronophagia and endothelial proliferation or necrosis. A marked degree of demyelination with preservation of neurons and their axons is considered to represent predominantly “Postinfections”, or an autoimmune encephalitis. The cerebral cortex especially the temporal lobe is often severely affected by HSV. The arboviruses tend to affect the entire brain. Rabies has a predilection for the basal structures.

Clinical – Manifestations

The progression and severity of disease are determined by the relative degree of meningeal and parenchymal involvement which in part is determined by the specific etiology. The clinical course resulting from infection with the same pathogen varies widely. Some children may appear to be mildly affected initially, only to lapse into coma and die suddenly. High fever ,violent convulsions interspread with bizarre movements and hallucination .

The onset of illness is generally acute although CNS signs and symptoms are often preceded by a nonspecific febrile illness of a few days duration. The presenting manifestations, in older children are headache and hyperesthesia and in infants irritability and lethargy. Headache is most often frontal or generalized adolescents frequently complain of retrobulbar pain, fever ,nausea , vomiting ,photophobia, and pain in the neck back, and legs are common.As body temperature increases there may be mental dullness, progressing to stupor in combination with bizarre movements and convulsions. Focal neurological signs may be stationary, progressive or fluctuating

Exanthems often preceede or accompany the CNS signs especially with echoviruses, coxsackieviruses, varicella zoster virus, measles, rubella and occasionally WNV. Examination often reveals nuchal rigidity without significant localizing neurologic changes.

Diagnosis

The diagnosis of viral encephalitis is usually made on the basis of the clinical presentation of nonspecificsymptoms. The CSF which usually shows a mild mononuclear predominance value in the evaluation of patients with suspected viral meningitis include on electro encephalogram EEG and neuroimaging studies. The EEG typically shows diffuse slow

wave activity usually without focal changes. Neuroimaging studies may show swelling of the brain parenchyma.

Laboratory Findings

The CSF contains from a few to several thousand cells per cubic millimeter. Early in the disease the cells are oftenpolymorphonuclear, later mononuclear cells predominate. This change in cellular type is often demonstrated in CSF samples obtained as little as 8-12 hr apart.

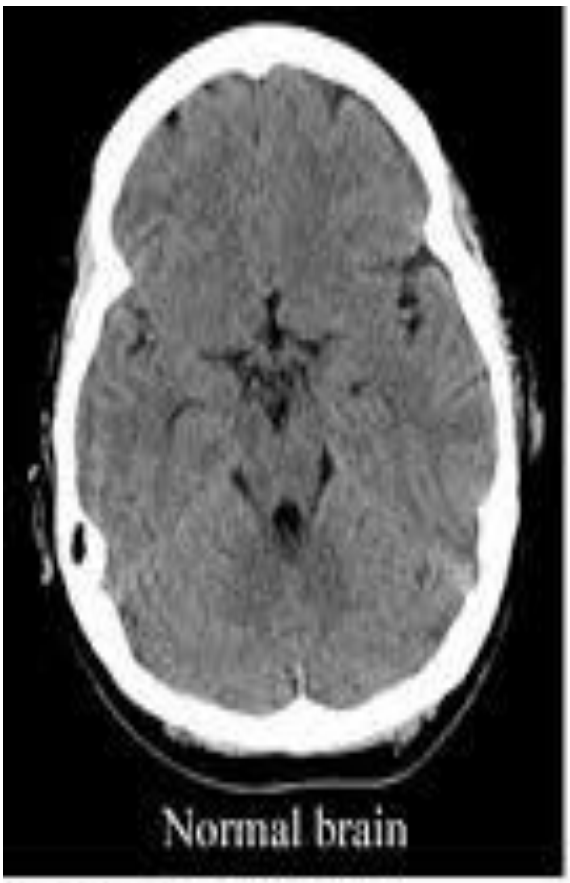


Figure 1

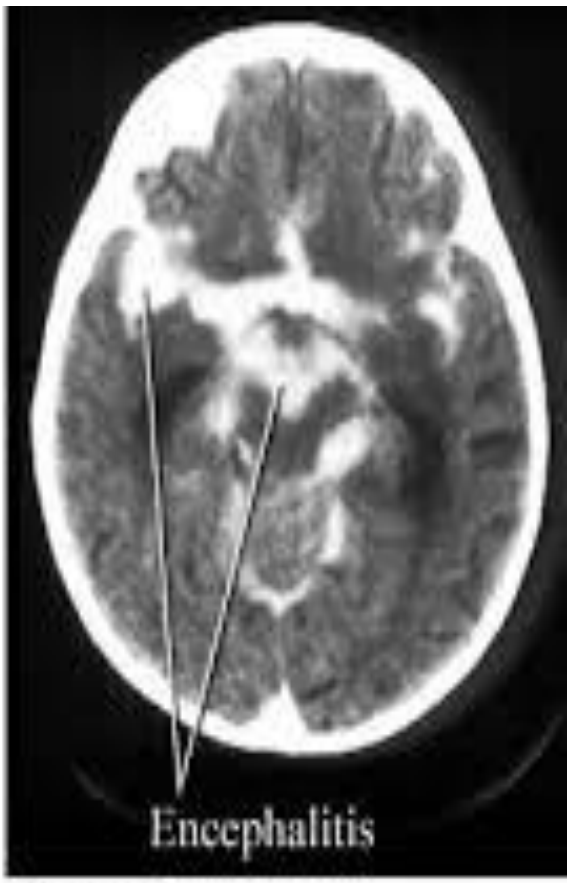
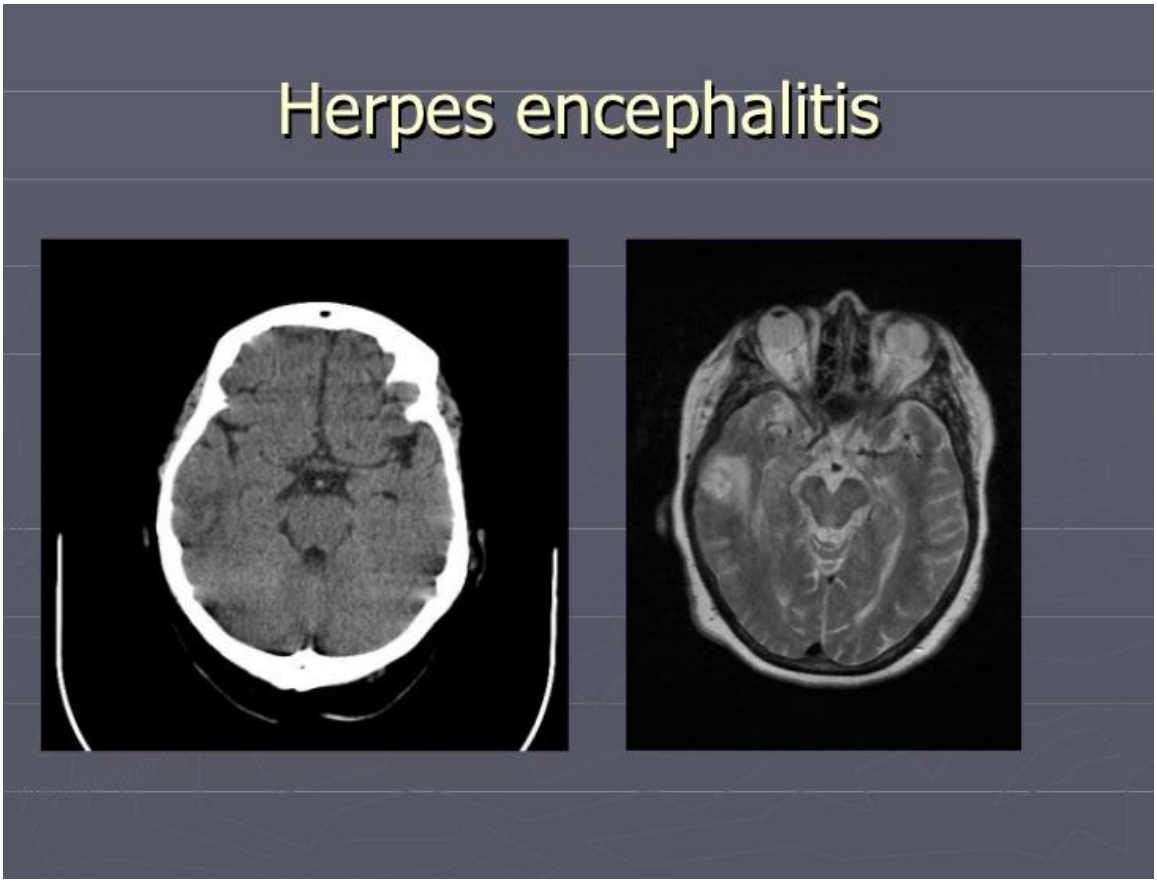


Figure 2



Treatment

Treatment of viral meningoencephalitis is mostly supportive. Treatment of mild disease may require only symptomatic relief .Headache and hyperesthesia are treated with rest, non aspirin containing analgesics, and a reduction in room light noise and visitors.

Acetaminophen is recommended for fever, opioid agents and medications to reduce nausea may be useful . Intravenous fluids are occasionally needed. More severe disease may require hospitalization and Intensive care.

Convulsions, cerebral edema ,inadequate respiratory exchange, disturbed fluid and electrolyte balance, aspiration and asphyxia increases ICP. Placement of a pressure transducer in the epidural space may be indicated. The risks of cardiac and respiratory failure or arrest are high with severe disease .

Prognosis

Supportive and rehabilitative efforts are very important after patients recover from the acute phase of illness. Motor in-coordination, convulsivedisorders, total or partial deafness and behavioral disturbances may follow viral CNS infections. Visual disturbances from chorioretinopathy and perceptual amblyopia may also occur. Neuro developmental and audiology evaluations should be part of the routine follow up of children who have recovered from viral meningo encephalitis.

Most children completely recover from viral infections of the NCS. Although the prognosis depends of the severity of the clinical illness and the specific causative organism.

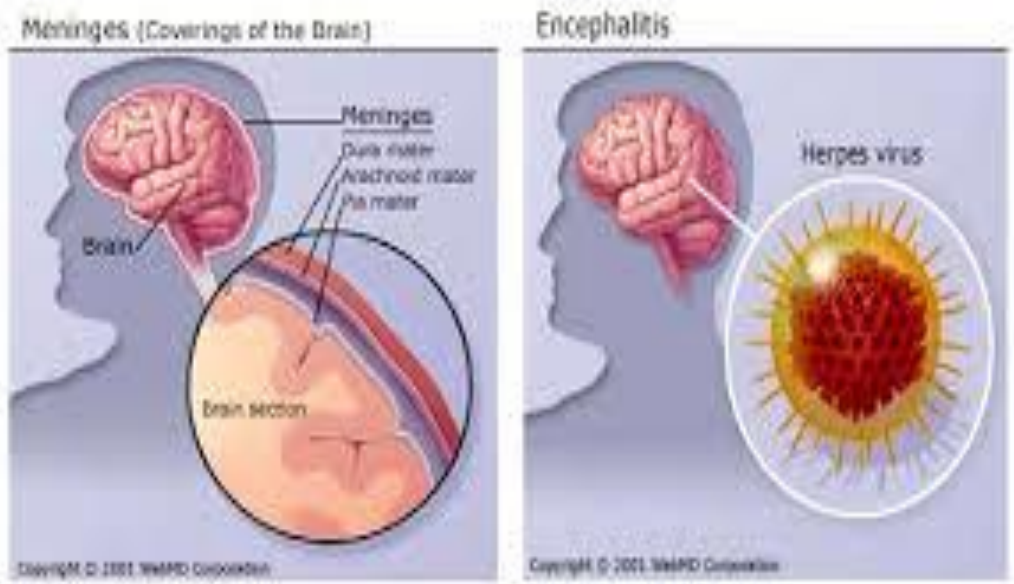
Prevention

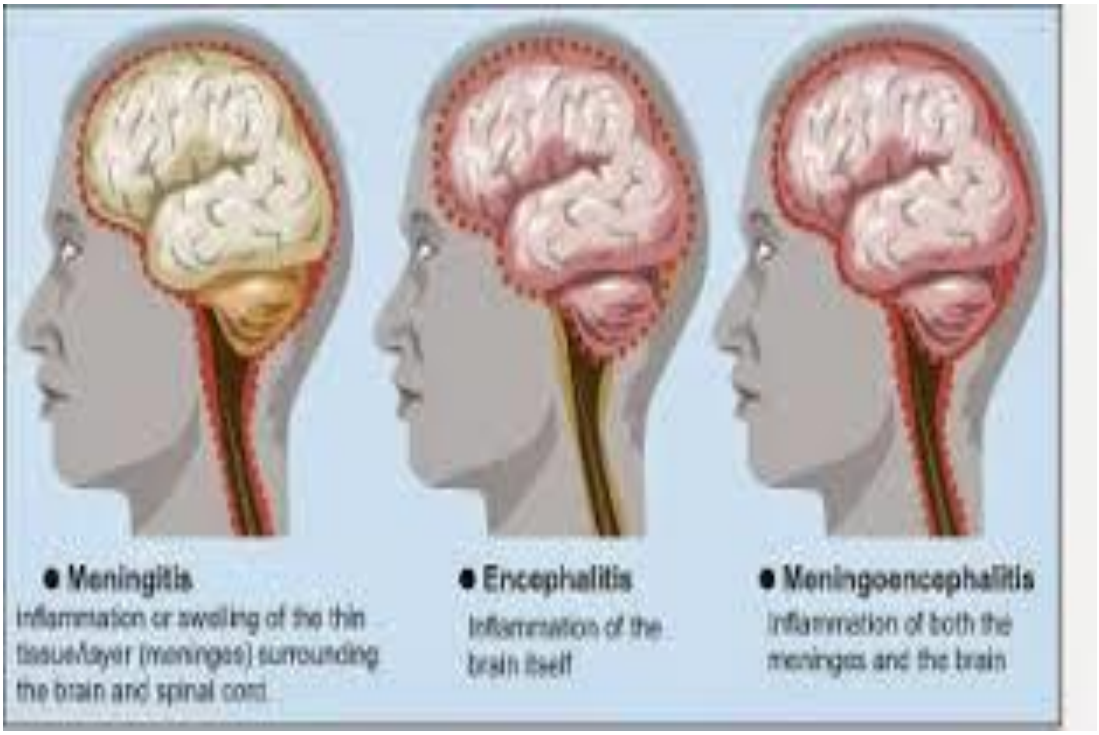
Widespread use of effective viral vaccines for polio, measles, mumps, rubella and varicella has almost eliminated CNS complications.

MENINGITIS
VERSUS
ENCEPHALITIS

ENCEPHALITIS	MENINGITIS
Encephalitis is an acute inflammation of the brain parenchyma	Meningitis is the inflammation of the protective layers of tissue or membranes covering the brain
Can be caused by Bacteria, viruses, and fungi	Commonly caused by viral agents
Exist only as a single form	Can occur as primary or secondary types
Symptoms include sudden fever, severe headache, nausea, vomiting, double vision, drowsiness, photophobia, skin discoloration and stiff neck	Symptoms include moderate-severe fever, seizures, behavioral changes, confusion, disorientation and related neurological signs
Diagnosed by routine blood examinations	Might require neuroimaging techniques along with blood tests
Will be given Ampicillin, combined with aminoglycoside or cephalosporin	Will be treated with intravenous Acyclovir for 10 days

Meningitis vs Encephalitis





Abhisheck Raj, Kalyanbratamandal et.al., They are study on clinico – epidemiological, etiological and imaging profile of acute viral meningoencephalitis in children age one month to 12 yrs, study at 'Calcuta Medical College, R.G. Kar Medical College, at Department of Pediatrics, Kolkata. The study is hospital based observation study in prospective manner. This study duration is 1 year totally 161 persons are included study. Mean average age was 5.2 ± 3.4 and Male : Female ratio of 1.8 : 1. Most common clinical presentation was fever with alternated sensorium. Out of 161 persons 48 cases are viral etiological and 130 cases are cause could not be find out.

CSF Cell count was 79% have lymphocytes predominate. All persons who are vitally stable MRI taken. 30% of children's have abnormal MRI finding. Out of 161 person 31 case died and 10 cases against medical advice and 112 cases of successfully discharge.

Farzaba.K. Beig, Abida Malik, et.al doing study on etiology and clinico - epidemiological profile of acute viral encephalitis in children of western uttarpradesh department of paediatrics, JawaharlNehur Medical Collage, Aligarh Musilm University College, eighty seven patients including in the study. Median age was 3.5 yrs Male Female ratio was 1.27:1 viral etiology was diagnosed in a total 19 cases among these the most common agent was EV-71 followed by measles agent

vericellaherpes simplex. Japanese encephalitis virus was not found in any study Enterovirus 71 infection caused significant morbidity in children mortality occurred in 50%. In their study generalized convulsions along with fever, altered sensorium were the significant findings in patients with viral encephalitis.

Dr. Mohan D Kashinkunti and Dr.Shiddappa et.al Doing study at department of pediatrics SDMCMS & Hospital Dharwad, tertiary care teaching hospital Karnataka. It is a prospective observation study over a period of 1 year in patients aged less than 15 yrs. They determine the possible etiology cerebrospinal fluid (CSF) analysis and imaging of brain was done. Totally hundred patients are studied among this viral encephalitis its most common etiology (35%) followed by Pyogenic meningitis, cerebral malaria (6%) and tuberculosis (5%) diagnosed and auto immune encephalitis (29%).

Dr. RitumoniSonowal, Dr.P.Dowerah et.al studied at department of pediatrics Assam Medical College about clinico-Aetiological study of acute bacterial meningitis in children. The study period was 1 year after taking a history and thorough clinical examination including lumbar puncture. Cases showing CSF findings were included in the study at and further evaluated. Totally 59 cases are include.

Totally 59 cases are include the study out of this 76.27% cases were below the age of 1 year and most frequent presenting features were fever (86.4%) altered sensorium (67%) convulsions (55.93%) CSF pressure was high in 50.84% cases CSF appearance was hazy in 44%. CSF protein ranged between 151-300mg/dl (69.49%) CSF cell count range (51 to 500) cells / cube millimeters.

Dr. All Khajeh, Dr. Batool Sharif – mood studied at Zahedan south eastern Iran Department of pediatrics Zehedan University of Medical Sciences. It is a current cross sections and descriptive. Study investigated the Medical records of the patients with meningo encephalitis from may 2010 to may 2014, then the patients were evaluated according to Sex, age clinical feature, risk factor, etiologic agents, acute or chronic form and clinical outcome. They 55% patients included their study among 55% patients (42%) girls and 58% boys) mean age of 85 months age range of 45 days to nine years 98% had acute meningo encephalitis out of 54 cases only 6 cases showed a positive test for etiology 48 patients had negative CSF culture the most common clinical symptom was fever (95%) and the least was seizure (16%). Mortality rate was 72% (four cases) There were no significant risk factors in children with illness, but there was a history of pulmonary TB in grandmother of the case with TB meningitis.

Dr.KalaYadhav me et.al studied at Department of pediatrics tertiary case hospital Bangalore Medical College totally 100 clinical suspected cases of 5 years of age included the study. To isolate identify and determine the antibiotic susceptibility patients of pathogens associated with bacterial Meningitis another aim is to comparatively evaluate of gram staining culture and bacterial antigen detection in CSF of the 100 cases 24 were diagnosed as acute bacterial meningitis (ABM) cases by Gram staining culture and latex agglutination test 21 (87.5%) cases were culture positive with 2 cases being positive for poly microbial isolates. Gram staining was positive in 17 (70.53%) cases and LAT was positive in 18 (33.33%) cases streptococcus pneumoniae was the predominant organism which was isolated and it was sensitive to antibiotics.

METHODOLOGY

MATERIALS AND METHODS

Types of Study

Descriptive study

Study Place

PICU (Paediatric Intensive Care Unit) in Government Medical College and Hospital, Coimbatore.

Study Population

Children in the age group of one month to 12 years who are admitted in PICU with symptoms ofmeningoencephalitis and also full fill the inclusive criteria during the study period of Nov 2016 to Aug 2017.

Study Period

Nov 2016 to Aug 2017 (10months)

Sample size

Non probable sample technique ,Time bound study (Nov 2016 to Aug 2017)

SELECTION CRITERIA

A. INCLUSION CRITERIA

All paediatric patients who got admitted in the PICU under the age group of one month to 12 years, with symptoms of Meningitis like fever, headache, vomiting, nuchal rigidity.

All paediatric patients who got admitted in PICU in the age group of one month to 12yrs, with symptoms of encephalitis like altered sensorium, seizures, focal neurological deficit.

B. EXCLUSION CRITERIA

1. Dyselectrolytemia
2. Cerebral malaria
3. Intra Cranial space occupying lesion (ICSOL)
4. “Reyes” syndrome
5. Enteric fever
6. Hepatic encephalopathy

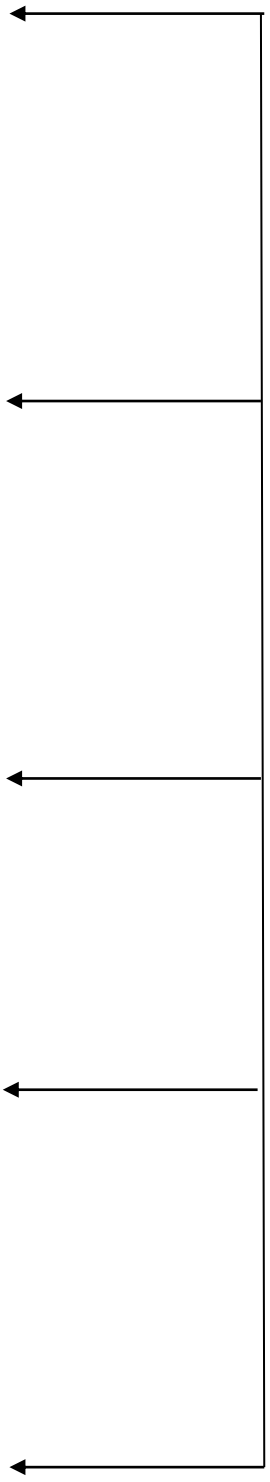
MAY 2016 MD joined

NOV 2016 EC Clearance

AUG 2017 Data Collection to be finished

SEP 2017 Follow Up

OCT 2017 to be submitted



MANEUVER

This study is undertaken in department of pediatrics, Government Coimbatore Medical College Tertiary Care Hospital. This is a hospital based descriptive observational study. It was conducted over a period of 10 month Nov-2016 to Aug- 2017. Institutional ethics committee approval was obtained before starting the study.

Study population consists of children aged one month to 12 years. Admitted with feature of Acute Meningo Encephalitis .Written consent was obtained from the parents (or) legal guardian of the eligible patient before recruitment in the study.

Clinical diagnosis of acute Meningo Encephalitis was made in the presence of following symptom.

- Fever
- Altered Sensorium

  With (or) without motor/ sensory defect
- Convulsion
- Head ache
- Vomiting

Child who presented with other cause like cerebral malaria, dyseletrolytemia, ICSOL (Intra Cranial space occupying

lesion),reyessyndrome, enteric fever, Hepatic Encephalopathy are excluded from this study.

All Peadiatric patients who will present to us with the feature of meningo encephalitis will be registered for this study. Clinical data such as Age, Sex, Resident Address, Developmental History, Immunisation Status, Exanthematous fever, Pre Hospital Treatment, Contact History of TB, MigrationalHistory, are enter in the Pre structural Profoma.

All Symptoms fever, head ache, vomiting, Seizure, altered sensorium, focal neurological deficit (FND) and pre hospital treatment like, Oxygen, IV. Fluids, Antibiotics, anti Viral areall documented.

Condition of the child arrival at the PICU, CMCH like AVPU Scale, ICP, GCS and seizure are recorded. Patient's details are kept in the ward.

Routine investigation such as complete haemogram, blood-sugar, serum electrolytes,renal function test, liver function test, peripheral smear for malarial parasite was done.

After full clinical examination and fundus examination, any contra indications for L.P (Lumbar Puncture) like increased ICP features, AF Bulg, 3^{r}or 6th nerve palsy, hypertension, bradycardia, severe

cardiopulmonary complications like shock, local skin infections should be ruled out.

Lumbar Puncture was done and CSF analysis like appearance, opening pressure ,cell count, protein and sugar, culture , sensitivity, Gene Expert are done.

Imaging MRI/CT and EEG done. Outcome complication like any hydrocephalus and other changes and hearing impairment (BERA) are recorded.

RESULTS

OBSERVATIONS AND RESULT

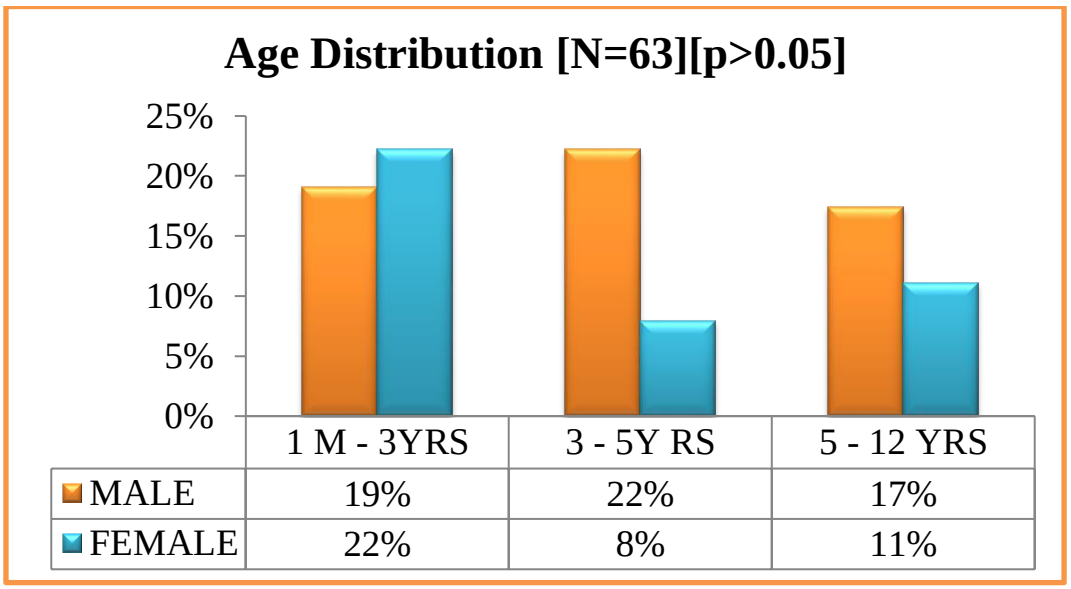
Our study is to assessclinico – epidemiological profile and outcome of meningoencephalitisin children aged one month to 12 yrs. in Coimbatore Medical College. We collected all data's and entered in the profoma for statistical analysis .Frequency are expressed in percentage. The difference in quantities variables groups were assessed by means of the unpaired test. The chi-square test was used to asses' differences in categorical variables between groups. A 'p' value of <0.05 using a two – tailed test was taken significant for all statistical tests. All data were analyzed with a statistical software package (SPSS. Version 16.0 for windows). Totally 63 patients who fulfilled the inclusions criteria were registered.

TABLE – 1

AGE DISTRIBUTION

AGE	MALE	(%)	FEMALE	(%)	TOTAL	(%)
1 M - 3YRS	12	19%	14	22%	26	41%
3 - 5Y RS	14	22%	5	8%	19	30%
5 - 12 YRS	11	17%	7	11%	18	29%
Total	37		26		63	

MEAN +/- SD	
MALE	4.81 +/-1.05
FEMALE	4.38+/-1.36
TOTAL	4.63+/-1.92

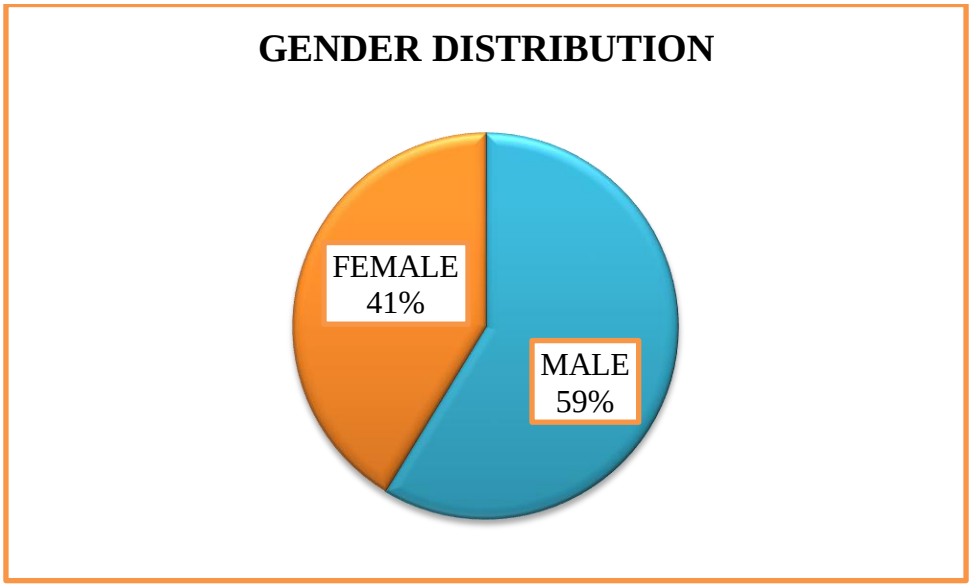


In our study out of 63 children, 41% (26 children) are agedbetween one month to 3yrs, 30% (19 children) are aged between 3 to 5yrs, and 29% (18 children) aged between 5 to 12yrs. Hence the most common age group is **one month to 3 years**.

TABLE – 2

GENDER DISTRIBUTION

GENDER	N	(%)
MALE	37	59%
FEMALE	26	41%
Total	63	

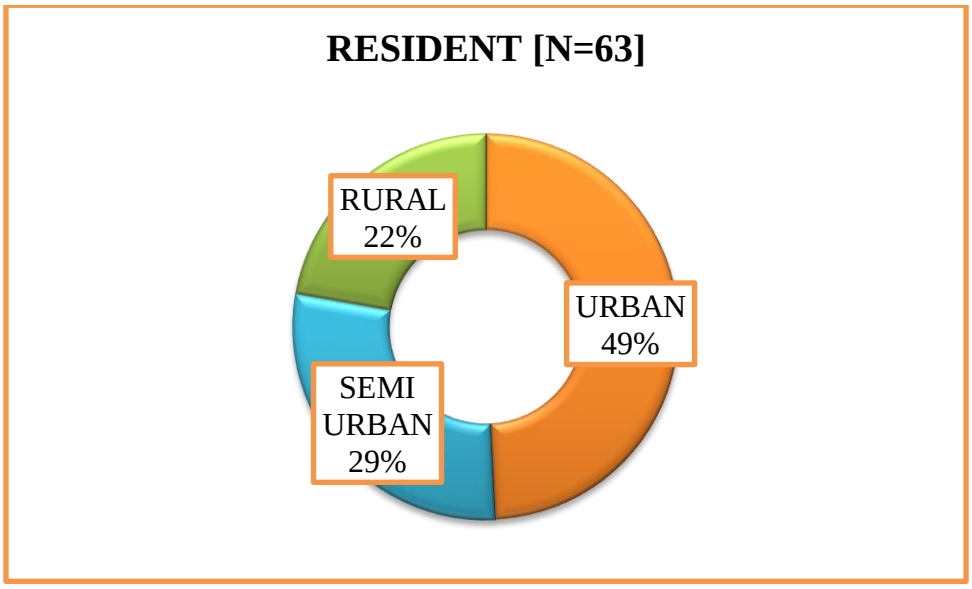


Out of 63 children's 59% (37 children) are Male and 41% (26 children) are Female. **Males are more commonly affected than Girls.**

TABLE – 3

RESIDENT

RESIDENT	N	(%)
URBAN	31	49%
SEMI URBAN	18	29%
RURAL	14	22%
Total	63	



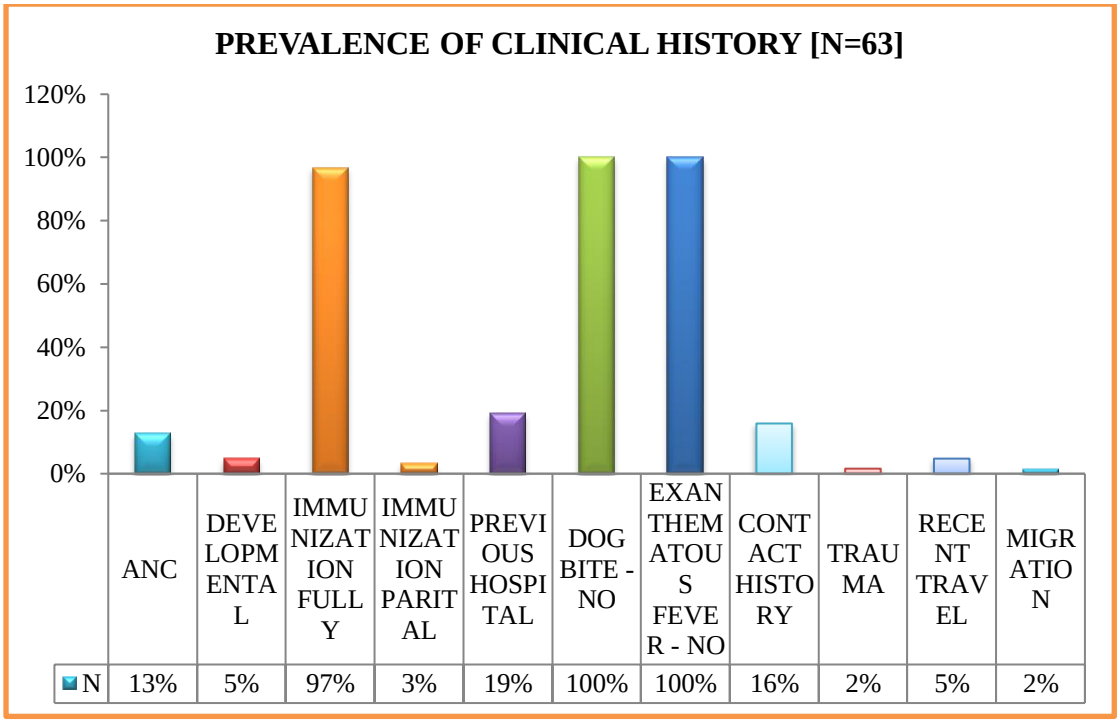
Regarding residence 49% (31 children) who are admitted from residence of urban area and 29% (18 children) are admitted from Semi Urban area and 22% (14 children) from rural area.

Urban children are more affected in meningo encephalitis.

TABLE – 4

CLINICAL HISTORY

PREVALENCE OF CLINICAL HISTORY	**N**	(%)
ANC ISSUES	8	13%
DEVELOPMENTAL HISTORY	3	5%
IMMUNIZATION STATUS - FULLY	61	97%
IMMUNIZATION STATUS - PARITAL	2	3%
PREVIOUS HOSPITAL ADMISSION	12	19%
DOG BITE HISTORY – NO	63	100%
EXANTHEMATOUS FEVER - NO	63	100%
CONTACT HISTORY	10	16%
TRAUMA HISTORY	1	2%
RECENT TRAVEL HISTORY	3	5%
MIGRATION HISTORY	1	2%

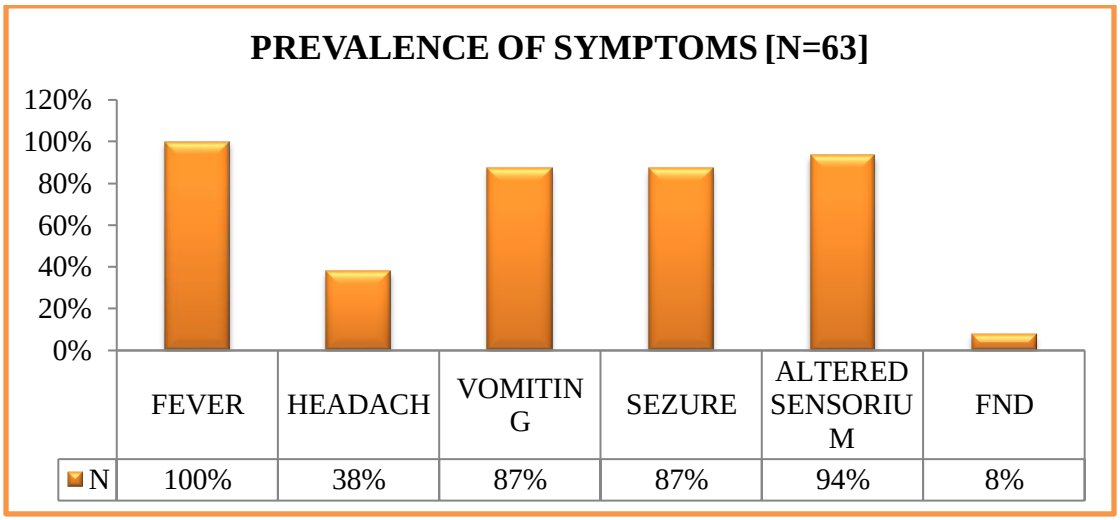


Regarding clinical history in our study 13% (8 children) have ANC issues, 5% (3 children) developmental delay, 3% (2 children) with partial immunization, 19%(12 cases) previous hospital admission, 16% (10 children) had contact history, 2% (1 children) trauma history and 5% (3 children) had recent travel history, none of the children have dog bit and exanthemata's fever.

TABLE – 5

SYMPTOMS

PREVALENCE OF SYMPTOMS	N	(%)
FEVER	63	100%
HEADACH	24	38%
VOMITING	55	87%
SEZURE	55	87%
ALTERED SENSORIUM	59	94%
FOCAL NEUROLOGICAL DEFICIT	5	8%

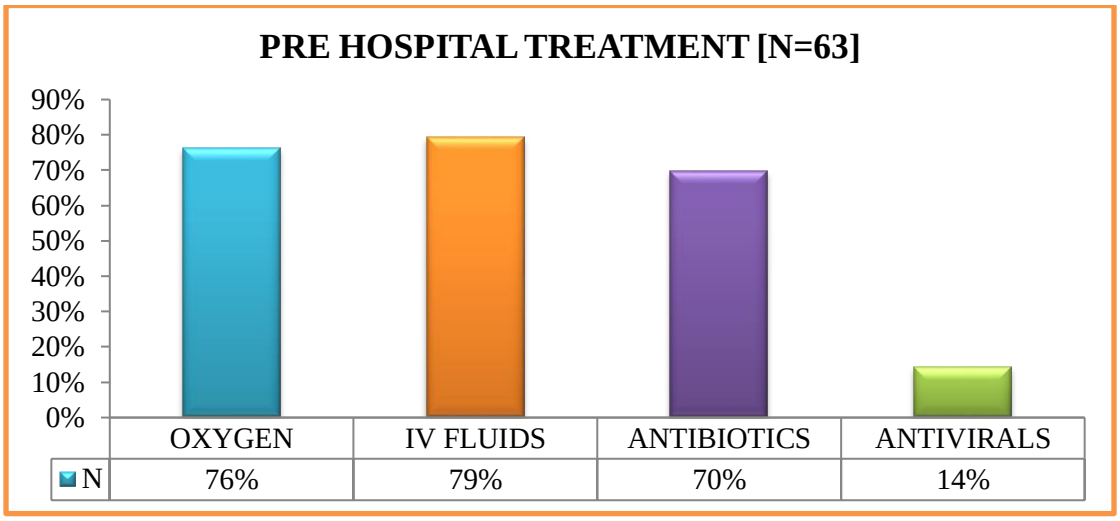


Out of 63 children all children (100%) had a fever, 94% (59 children) had an altered sensorium, 87% (55 children) had seizures and vomiting, headache 38% (24 children) headache and only 8% (5 children) focal neurological deficit. The most common symptoms in our study is **fever and altered sensorium.**

TABLE – 6

PRE HOSPITAL TREATMENT

TREATMENT	N	(%)
OXYGEN	48	76%
IV FLUIDS	50	79%
ANTIBIOTICS	44	70%
ANTIVIRALS	9	14%

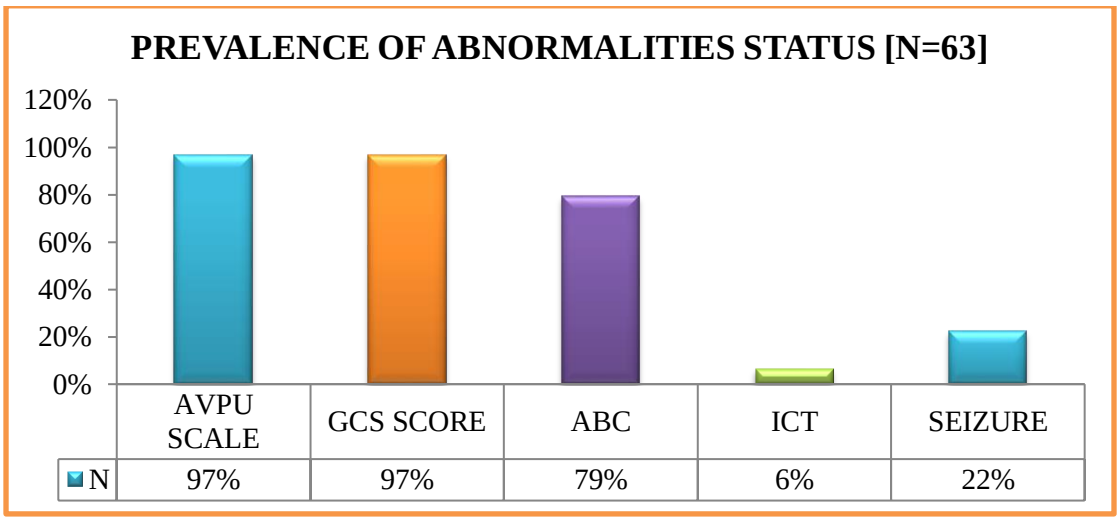


Regarding Pre Hospital Treatment 79% (50 children) had IV fluids, 76% (48 children) oxygen therapy, 70% (44 children) antibiotic therapy and only 14% (9children) antiviral therapy. The most common pre hospital treatment is **IV fluids and oxygen therapy**.

TABLE – 7

AT PICU ADMISSION

PREVALENCE OF ABNORMALITIES STATUS	N	(%)
AVPU SCALE	61	97%
GCS SCORE	61	97%
ABC	50	79%
ICT	4	6%
SEIZURE	14	22%

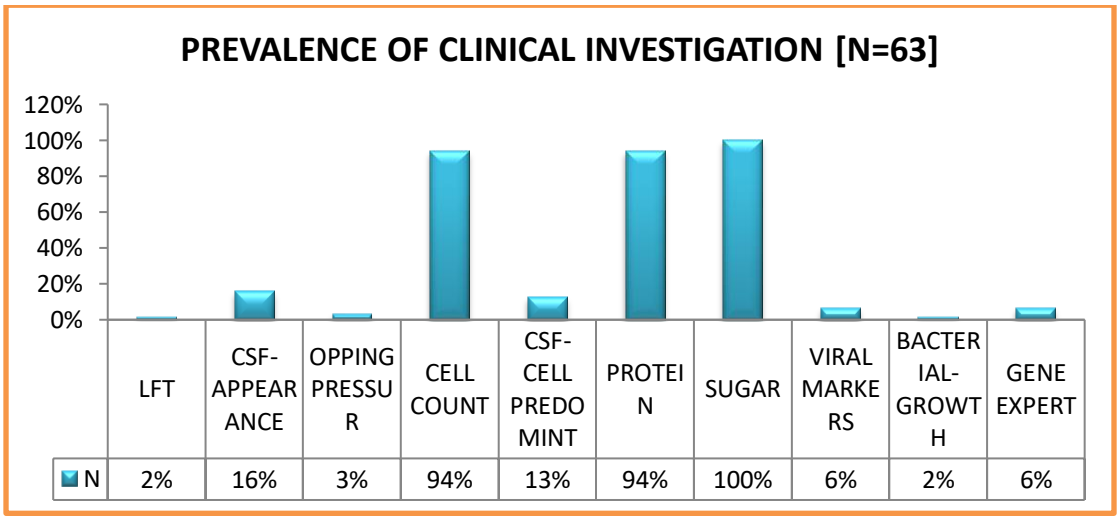


At the time of admission in PICU 97% (61 children) had abnormal AVPU scale and GCS score, 79% (50 children) abnormal ABC Status, 22% (14 children) seizure activity at the time of admission and only 6% (4 children) increased ICP. AVPU and GCS score were the most common abnormal symptoms at the PICU presentation

TABLE – 8

CLINICAL INVESTIGATION

PREVALENCE OF CLINICAL INVESTIGATION	N	(%)
LFT	1	2%
CSF-APPEARANCE	10	16%
OPPING PRESSUR	2	3%
CELL COUNT	59	94%
CSF-CELL PREDOMINT	8	13%
PROTEIN	59	94%
SUGAR	63	100%
VIRAL MARKERS	4	6%
BACTERIAL-GROWTH	1	2%
GENE EXPERT	4	6%

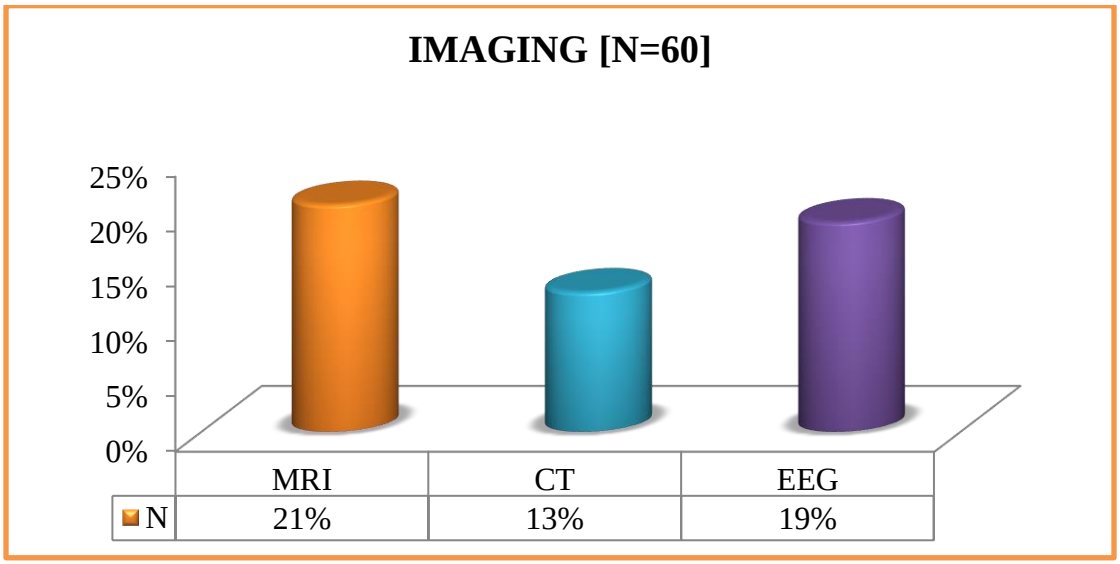


Regarding clinical investigation 94% (59 children) have increased cell count and elevated CSF protein, 16% (10 children) cloudy CSF appearance, 13% (8 children) had CSF PMN predominate. 6% (4 children) viral marker of dengue encephalitis (2) Japanese encephalitis, 6% (4children) positive gene expert – TB Meningitis, 2% (1children) bacterial growth (St Pneumonia), 3% (2children) increased opening pressure when doing CSF Analysis.

TABLE – 9

IMAGING

IMAGING	N	(%)
MRI	13	21%
CT	8	13%
EEG	12	19%

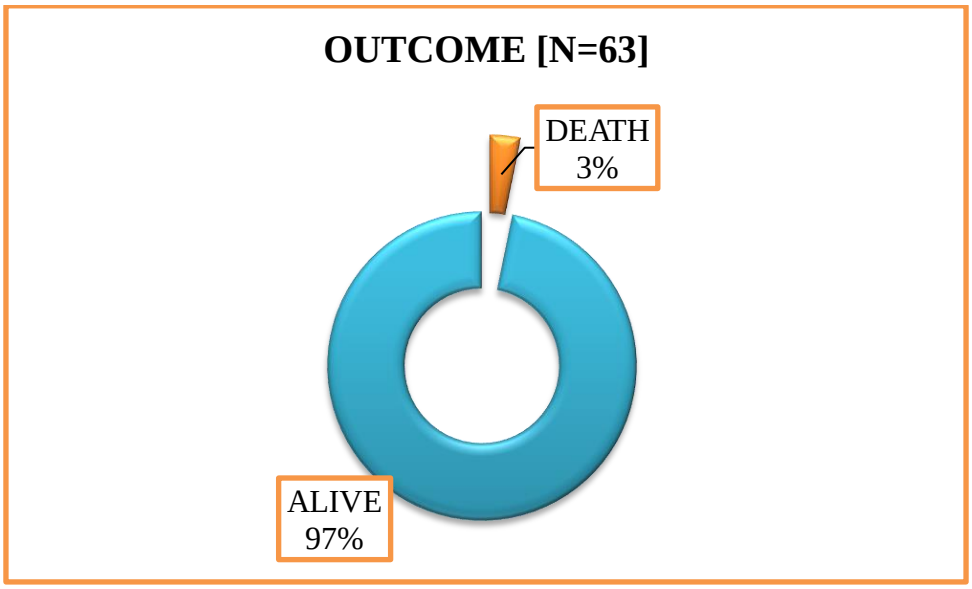


Out of 63 children 21% (13children) had abnormal MRI and 13% (8children) abnormal CT, 19% (12children) abnormal EEG pattern.

TABLE – 10

OUT COME

OUTCOME	N	(%)
DEATH	2	3%
ALIVE	61	97%
Total	63	

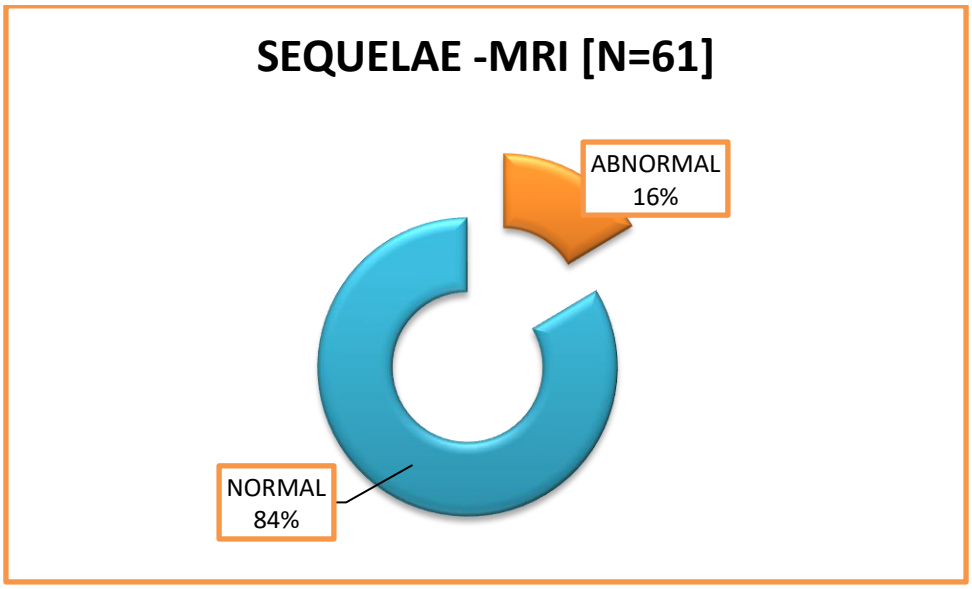


Out of 63 children's 97% (61 children) have alive and 3% (2children) death.

TABLE – 11

SEQUELAE MRI

SEQUELAE -MRI	N	(%)
ABNORMAL	10	16%
NORMAL	51	84%
Total	61	

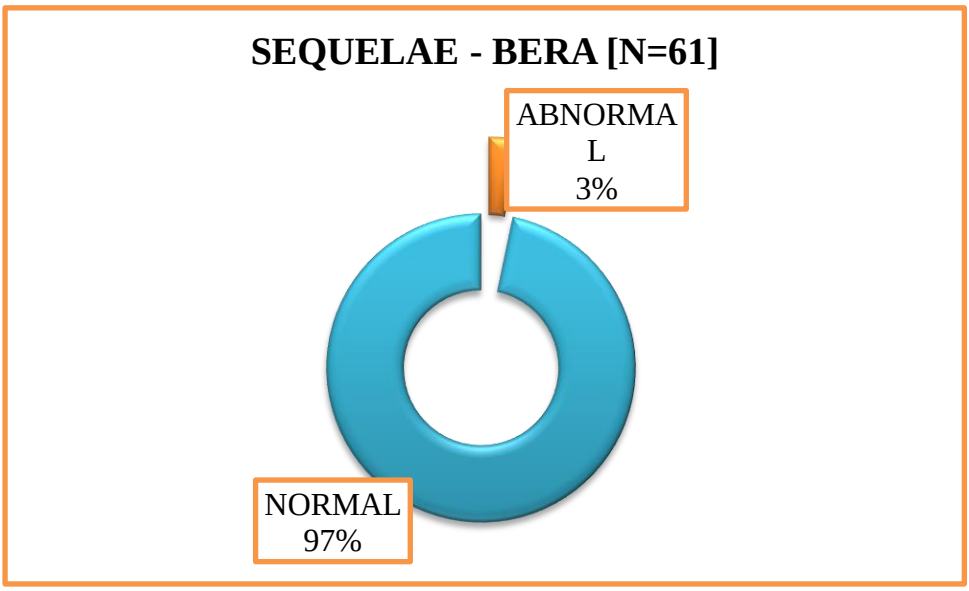


After one month of discharge followed MR / CT scan done only 16% (10 children) abnormal MRI finding, 84% (51children) normal MRI without any neurological damage.

TABLE – 12

SEQUELAE BERA

SEQUELAE -MRI	N	(%)
ABNORMAL	2	3%
NORMAL	59	97%
Total	61	



After one month of discharge we do a hearing evolution by BERA only 3% (2children had abnormal sensory neural hearing loss and 97% (59children) normal hearing.

TABLE – 13

ASSOCIATION OF CLINICAL VARIABLES WITH OUT COME [N=63]

OUT COME	DEATH	(%)	ALIVE	(%)	Total	Sig
CSF APPEARANCE						
NORMAL	0	0%	53	100%	53	<0.05
CLOUDY	2	20%	8	80%	10	
OPPING PRESSUR						
INCREASED	1	50%	1	50%	2	
NORMAL	1	2%	60	98%	61	<0.05
CELL COUNT						
NORMAL	0	0%	2	100%	2	
INCREASED	2	3%	57	97%	59	>0.05
3	0	0%	2	100%	2	
CSF-CELL PREDOMINT						
NORMAL	0	0%	55	100%	55	
INCREASED	2	25%	6	75%	8	<0.05
PROTEIN						
NORMAL	0	0%	4	100%	4	
INCREASED	2	3%	57	97%	59	>0.05
SUGAR						
INCREASED	2	3%	61	97%	63	
VIRAL MARKERS						
NO	2	3%	57	97%	59	
YES	0	0%	4	100%	4	>0.05
BACTERIAL-GROWTH						
NO	1	2%	61	98%	62	
YES	1	100%	0	0%	1	<0.05
GENE EXPERT						
NO	1	2%	58	98%	59	
YES	1	25%	3	75%	4	<0.05

In children whose CSF appearance was cloudy at the time of Initial examination highly correlated with mortality in our study (20%)

Increased Mortality in these babies may be probably due to increased virulence of the offending organism and uninhibited proliferation in Brain.

Increased opening pressure at the time of CSF examination was also a major factor increasing the rate of mortality among the children with meningo encephalitis.

In our study 50% of babies with increased opening pressure resulted in Mortality. Increased ICP, 2^0 to diffuse cerebral edema and blockage of ventricular system is highly correlative with prognosis in children with meningo encephalitis.

In our study only 3% of the babies had increased cell count. This is not statistically associated with mortality. Because most of the children had already got treatment with some antibiotic and antiviral drugs before admittingto our hospital.

In our study children whose CSF cell count were predominantly PMN (25%) highly correlated with the mortality

With significant the p value of P <0.05. Even though these children were treated prior to admission to hospital they had increased mortality.

It shows that treatment based on the sensitivity of the organisms and adequate dose of antibiotics is very important to decrease the mortality of the children.

Regarding CSF protein in our study shows only 3% are increase the mortality associate with increased protein in the CSF. It is not statistical significant p value >0.05.

In our study, CSF analysis of viral marker and mortality association not correlated p value is not significant. This is why because in our hospital viral etiologic agents are diagnosed by culture and sensitive and pattern growth.

We are not using PCR method and IgM Eliza to detect the viral marker. When doing the culture in the growth of the organism in CSF is yielding power is very low.

When Bacterial growth in CSF and mortality association is 100%. It is highly correlated with bacteria growth in CSF culture with very high mortality.

Child who had CSF bacteria growth of positive high significant correlated with mortality. it is very good prognostic Indicator of outcome following sequelae in the children.

It shows even the children got antibiotic therapy in Pre hospital Treatment mortality increase due to highly virulent of organism and poor Immune status of the children and age and epidemiological back ground also important.

In CSF Gene expert and outcome associates shows 25% children's who had TB positive children highly correlate with mortality.

In our hospital CSF Gene expert shows Tuberculosis meningitis positive if detect by CSF PCR method. It shows Tuberculosis meningitis in developing country like India show major mortality and morbidity in associate with the outcome of meningoencephalitis in children age one month 12 years.

TABLE – 14

COMPARSION OF CLINICAL VARIABLES WITH SEQUELAE MRI [N=61]

SEQUELAE MRI	ABNORMAL	(%)	NORMAL	(%)	Total	Sig
CSF APPEARANCE						
NORMAL	6	11%	47	89%	53	<0.05
CLOUDY	4	50%	4	50%	8	
OPPING PRESSUR						
INCREASED	1	100%	0	0%	1	
NORMAL	9	15%	51	85%	60	<0.05
CELL COUNT						
NORMAL	0	0%	2	100%	2	
INCREASED	8	14%	49	86%	57	<0.05
3	2	3%	0	0%	2	
CSF-CELL PREDOMINT						
NORMAL	8	15%	47	85%	55	
INCREASED	2	33%	4	67%	6	>0.05
PROTEIN						
NORMAL	2	50%	2	50%	4	
INCREASED	8	14%	49	86%	57	>0.05
SUGAR						
INCREASED	10	16%	51	84%	61	
VIRAL MARKERS						
NO	8	14%	49	86%	57	
YES	2	50%	2	50%	4	>0.05
BACTERIAL-GROWTH						
NO	10	16%	51	84%	61	
GENE EXPERT						
NO	8	14%	50	86%	58	
YES	2	67%	1	33%	3	<0.05

Once children's are alive after the full course of antibiotic, antiviral and other supportive measures given then successfully discharged.

After One month of discharged we review the children and clinically examination, any neurological deficits, development and follow up MRI or CT taken to ruled out the any complication of meningo encephalitis. We also doing hearing evaluation by BERA. Then we compare association between the initial CSF parameters and one month follow up MRI or CT and hearing abnormality by BERA.

In our study children where CSF appear was cloudy at the time of initial CSF examination highly correlated with follow up of MRI or CT abnormally in about 50%.

Increased morbidity in this baby is may be due to increased virulence of the offending agents and its un inhibited proliferation in the brain.

CSF opening pressure at the time of initial CSF exam those who are increase is a major factor increased rate of morbidity among the children with meningo encephalitis.

In our study 100% of baby with increased opening pressure follow up MRI or CT shows the abnormal finding like

CT - Meningeal Enhancement, perivascular inflammation, Thrombosis in Transverse Sinus, Sagittal Sinus, Vasculitis and hydrocephalous.

MRI - Parenchymal lesion, celebrities, ependymal Enhancement, subdural empyema

It is highly correlated with the increases, mortality and significant ‘p’ value less than <0.05%

In follow up children who have increased the cell count in CSF 40% of them shows the MRI abnormality this is also shows as a p value of <0.05 highly correlate units children who have meningo encephalitis. When the cell count increase it represent the active growth up the microbial agents present in CSF. Even though antibiotic and antiviral are given, who are increase the cell count they are more prone for follow up complication in MRI / CT Change.

Children who had increased cell count with lymphocyte predominate present in the initial CSF examination shows after one month follow up among that 14% children have abnormal MRI change.

It is not significant correlated with MRI abnormality p value is >0.05. It is probably due to antibiotic and antiviral therapy given prior to admission in our hospital.

CSF protein value and follow up MRI changes after one month shows only 14% of mortality it's not statistically significant correlated in our study the 'p' value is < 0.05.

Viral marker present in CSF Initial examination and one month follow MRI abnormality is 14%. It is not significant correlated because viral culture and sensitivity yielding power is very low in our hospital study.

Gene expert in initial CSF examination and one month follow up discharge MRI / CT change shows 67% of children's have abnormality. It is highly correlated the morbidity of the children's who are present with acute meningo encephalitis.

In India like developing country TB is more common organism give the meningitis sequelae. Even though earlier ATT started some of the complications are not preventable. Only BCG Immunizations – 0 dose 100% fully covering reduce the problem.

TABLE – 15

COMPARSION OF CLINICAL VARIABLES WITH SEQUELAE BERA [N=61]

SEQUELAE MRI	ABNORMAL	(%)	NORMAL	(%)	Total	Sig
CSF APPEARANCE						
NORMAL	1	2%	52	98%	53	>0.05
CLOUDY	1	13%	7	88%	8	
OPPING PRESSUR						
INCREASED	1	100%	0	0%	1	
NORMAL	1	2%	59	98%	60	<0.05
CELL COUNT						
NORMAL	0	0%	2	100%	2	
INCREASED	8	13%	55	87%	63	>0.05
3	0	0%	2	100%	2	
CSF-CELL PREDOMINT						
NORMAL	1	2%	54	98%	55	
INCREASED	1	17%	5	83%	6	<0.05
PROTEIN						
NORMAL	0	0%	4	100%	4	
INCREASED	2	4%	55	96%	57	>0.05
SUGAR						
INCREASED	2	3%	59	97%	61	
VIRAL MARKERS						
NO	1	2%	56	98%	57	
YES	1	25%	3	75%	4	<0.05
BACTERIAL-GROWTH						
NO	2	3%	59	97%	61	
GENE EXPERT						
NO	2	3%	56	97%	58	
YES	0	0%	3	100%	3	>0.05

We compare the initial CSF analysis and abnormal changes in hearing evolution by BERA after one month of discharge of the Children.

CSF who have cloudy BERA is abnormal at the end of one month by 30% it is statistical not significant and not correlated in our study ‘p’ value is less than 0.05.

Children who have increased CSF opening pressure in the initial CSF examination shows increased the abnormality of hearing (sensory Neural Deafness) by BERA of 100%. It shows increase opening pressure, increase the morbidity is highly correlated with increase the hearing loss ‘p’ value significantly low < 0.05. It shows the increase opening pressure damaged the inner hair cell and produce (Sensory Neural Deafness).

Initial increase the cell count of CSF shows only 13% of abnormality to not significant increased morbidity not correlated in our study ‘p’ value is significant this >0.05.

CSF cell count whom had increased polymorph predominate initial CSF examination and abnormal hearing impairment by BERA is 17% shows highly correlated with morbidity in our study statistical significant ‘p’ value of < 0.05 and polymorphic lymphocytes early damage to the inner hair cells and produce the significant abnormality.

Increase CSF protein in the initial CSF examination and abnormal BERA shows only 4%. It is not statistical significant ‘P’ value of > 0.05.

It our study CSF viral marker positivity in the initial CSF examination and after one month of follow up BERA hearing abnormality is 25%.

It shows very highly correlated with viral marker positivity children’s had hearing impairment. Even though stating of antibacterial and antiviral therapy organism enter and damage to the inner hair cells earlier. It is highly correlated with morbidity statistical significant ‘p’ value of < 0.05.

Gene expert in the initial CSF examination and after one month of BERA shows no hearing impairment ‘P’ value is > 0.05 is statistically not significant.

DISCUSSION

DISCUSSION

This study was conducted to study the clinico-epidemiological profile and outcome of acute meningoencephalitis in children aged one month to 12 years and the association between the CSF analysis and outcome, complication of acute meningoencephalitis.

In our study about Gender distribution males are more common than females with Males 59% (37 children) and Females 41% (26 Children). **Abhishek Roy**et.al., conducted in west Bengal and **FarzanaK.Beig** conducted at Uttar Pradesh, both of the above studies support my study which also have a male predominance. The male predominance may be due to outdoor life activity, playing and agricultural filed work.

Age distribution one month to 3yrs age are more common age 41% (26 children) 3 - 5 yrs 30% (19 children) 5 – 12yrs 29% (18 children) **Dr. RitumoniSonowal** et.al., conductedin the department of pediatrics Assam Medical College, **Abhishek Roy** et.al., conducted in West Bengal showed the same age distribution which supports our study.

In our study regarding residence , Urban childrenare most commonly affected 49% (31 children), Semi Urban 29% (18 Children)

and Rural 22% (14 Children). So **most commonly urban children are affected by meningoencephalitis.**

Clinical History about ANC issue prevalence is 30% (8 children) ,developmental delay 5% (3 children), partially immunized 3% (2children), previous hospital admission 19% (12 children), previous conduct history 16% (10 children), trauma history 2% (1 children) and recent travel history 5% (3 children).

In our study clinical symptoms at the time of admission fever is 100% (63children), altered sensorium 94% (59 children),vomiting and seizure 87% (55 children), headache 38% (24 children), FND only 8% (5 children). So **most common symptom at the time admission fever and alter sensorium.**

Abhishek Royet.al., conducteda study in west Bengal and **FarzanaK.Beig** conducted a study at Uttar Pradesh, both of the above studies support my study. They also have a **most common symptoms is fever and alter sensorium.**

Before arrival at our hospital 76% (48 Children) were treated with oxygen therapy, 79% (50 children) had IV fluids and 70% (44 children)hadbeen treated with antibiotics only 14% (9 children). So pre **hospital treatment with IV fluid therapy and oxygen therapy was**

received by most of the children. Antiviral therapy was the least that was given to the children. Most common etiological organisms are viral but most of the childrenhad not received early antiviral coverage.

When children were admitted at PICU, AVPU Scale, GCS, ABC and increased ICP, seizure activity were recorded. In our study 97% (61children) have AVPU Scale and low GCS score 79% (50 children) ABC abnormality 22% (14 children) have seizure at the time admission. Only 6% (4 children) had increased ICP. So **most common abnormal finding when get in PICU admission is low GCS and AVPU Scale abnormality. Least is ICP seizure.**

Regarding clinical investigation CSF appears cloudy in 67% (10 children), increased opening pressure 3% (2children), cell count predominance 94% (59 children). **Dr. RitumoniSonowal**et.al., conducted in the department of pediatrics, Assam Medical College support our study similar value they also had.

Viral markers 6% (4 children) and bacterial growth 2 % (1 child) **Abhishek Roy** et.al., conducted in west Bengal and **FarzanaK.Beig** conduct at Uttar Pradesh, both of the above studies did not support my study because bacteria and viral identification was done by CSF culture and sensitivity and growth of the acteria by culture plate. PCR method

or IgM Elisa was not used in this study . Compared to PCR and IgM Elisa, yielding power of the culture and sensitivity is low.

Regarding imaging abnormity MRI had 21% (13 children), CT had 13% (8 children), EEG abnormality 19% (12children) in children of meningoencephalitis. **Abhishek Roy**et.al., conducted in west Bengal , support our study.

Out of 63 children 2 children died(3% death), 61 children(97%) are alive. Death rate is decreased compared to **Abhishek Roy**et.al., conducted in west Bengal and **FarzanaK.Beig** conducted at Uttar Pradesh. This is due to early referral to higher tertiary centres and appropriate antibiotic, antiviral and anti edema measures given to reduce mortality.

SUMMARY

SUMMARY

Total no.of cases studied 63.

- **Age Distribution**

1 Month to 3 Yrs	:	41% (26 Children)
3 Yrs to 5 Yrs	:	30% (19 Children)
5 Yrs to 12 Yrs	:	29% (18 Children)

- **Gender Distribution**

Male	:	59% (37 Children)
Female	:	41% (21 Children)

- **Resident**

Urban	:	49 % (31 Children)
Semi Urban	:	29% (18 Children)
Rural	:	22% (14 Children)

- **Prevalence of Clinical History**

ANC Issue	:	13% (8 Children)
Developmental delay History	:	5% (3 Children)
Immunization Status fully:		97 % (61 Children)
Immunization Status Partially	:	3% (2 Children)

Previous Hospital Admission	:	19% (12 Children)
Contact History	:	16% (10Children)
Trauma History	:	2% (1Children)
Recent Travel History	:	5% (3 Children)
Migration History	:	2% (1Children)

- **Prevalence of Symptoms**

Fever	:	100% (63 Children)
Altered Sensorium	:	94% (59Children)
Vomiting	:	87% (55 Children)
Seizure	:	87% (55 Children)
Headache	:	38% (24Children)
FNT	:	8% (5 Children)

- **Pre Hospital Treatment**

IV Fluids	:	79% (50Children)
Oxygen	:	76% (48Children)
Antibiotic	:	70% (44Children)
Antiviral	:	14% (9Children)

- **AT PICU Admission**

AVPC Scale	:	97% (61Children)
GCS Score	:	97% (61Children)
ABC Abnormal	:	79% (50Children)
ICP	:	6% (4Children)
Seizure	:	22% (14Children)

- **Investigations**

CSF Appearance	:	16% (10Children)
CSF Opening pressure	:	3% (2Children)
CSF Cell count	:	94% (59Children)
CSF Cell predominance	:	13%(8Children)
CSF Protein	:	94% (59Children)
CSF Sugar	:	100% (63Children)
CSF Viral Marker	:	6% (4Children)
CSF Bacterial Growth	:	2% (1Children)
CSF Gene Expert	:	6% (4Children)

- **Imaging**

MRI	:	21% (13Children)
CT	:	13% (8Children)
EEG	:	19% (12Children)

- **Outcome**

Death	:	3% (2Children)
Alive	:	97% (61Children)

- **Sequelae -MRI**

Abnormal	:	16% (10Children)
Normal	:	84% (51Children)

- **Sequelae BERA**

Abnormal	:	3% (2Children)
Normal	:	97% (59Children)

Regarding association between initial CSF analysis VS Clinical outcome CSF appearance, opening pressure, CSF cell predominant, CSF bacterial growth, CSF Gene-expert give a highly correlated and significant 'p' value of < 0.05 others like CSF cell count, CSF protein, CSF Viral market not correlated and 'p' value is > 0.05.

Regarding initial CSF analysis VS follow up MRI shows CSF appearance, opening pressure, CSF cell count and CSF Gene-expert give a highly correlated and significant 'p' value of < 0.05 others like CSF cell predominant, CSF protein, CSF Viral market not correlated and 'p' value is > 0.05.

Association of CSF analysis and hearing defects on follow up.

Patients with high CSF opening pressure, CSF cell predominance and positive CSF viral markerswere found to have more hearing defects in BERA on follow up (p value of <0.05).

CSF appearance, CSF total cell count elevation, CSF protein, Gene expert analysis, CSF Viral markers were found to have no correlation with hearing defect(‘p’ value > 0.05).

CONCLUSION

CONCLUSION

- **Predominant age group involved was between 1 month to 3 year, which indicates the immaturity of Blood Brain Barrier.**

- **Viral infections were the most common etiology. Dengue virus followed by Japanese Encephalitis.**

- **CSF opening pressure and gross physical abnormality acts as an important prognostic indicator for outcome in children with acute CNS infection.**

- **Poor GCS and resistant seizures indicated the grave prognosis.**

LIMITATIONS OF STUDY

LIMITATIONS OF THE STUDY

Most common etiology of meningoencephalitisis viral in origin.But for detection of the viruses IgMELISA, Nuclear Method and PCR are the investigations of choice ;that has a high yielding power .CSF culture technique that has been used in this study has a comparatively low yield for detecting viruses.

Auto immune encephalitis is one of the most common etiology of acute meningoencephalitis, which is not included in this study due to resource limitations.

In this study patients had been under follow up for a period of one month after which hearing evaluation andneuro imaging has been done.

But for detecting meningoencephaliticsequelae like neurological defects,hearingdefects,visual abnormalities and psychosocial behavioral disorders a long term follow up is needed.

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ANNEXURE

xg;g[jy; gotk;

bgah] :

taJ :

ghypdk; :

Kfthp :

nfhit muR kUj;Jtf; fy;Y}hp kUj;Jtkidapy; kUj;Jth;. **kU.eh.rrpFkhh**; jiyikapy; eilbgWk; \isf;fha;r;ry;/ mjd; tpist[fs; kw;Wk; kWghpnrhjid cs;spl;l Ma;tpy; KGrk;kjj;Jld; fye;Jf; bfhs;fpnwd;. ,e;j Ma;tpy; vd;id / vd; FHe;ij gw;wpa midj;J tptu';fis bjhptpg;ngd; vd;Wk;/ me;j tptu';fis ghJfhg;g[ld; ,e;j Ma;tpy; btspapl Ml;nrgid ,y;iy vd;W bjhptpj;Jf; bfhs;fpnwd;;. ve;j neuj;jpYk; Ma;tpy; ,Ue;J tpyf;fp bfhs;Sk; chpik czL vd;W mwpntd;.

,lk; :

njjp :

ACUTE MENINGO ENCEPHALITIS-

CLINICAL PROFILE AND OUTCOME

S.NO. :

NAME :

AGE
:

1 MONTH TO3 Yrs.	3 TO 5Yrs	5 TO 12Yrs

SEX : MALE / FEMALE

RESIDENTIAL ADDRESS :URBAN / SEMI URMAN / RURAL

ANC ISSUES : YES / NO

DEVELOPMENTAL H/O :YES / NO

IMMUNISATION STATUS : FULLY/PARTIAL/NILL

PREVIOUS HOSPITAL ADMISSION :YES / NO

DOG BITE H/O :YES / NO

EXANTHEMATOUS FEVER IF ANY :YES / NO

CONTACT H/O TB :YES / NO

TRAUMA H/O :YES / NO

RECENT TRAVEL H/O :YES / NO

MIGRATION H/O :YES / NO

SYMPTOMS

	YES	NO
FEVER		
HEADACHE		
VOMITING		
SEIZURE		
ALTERED SENSORIUM		
FOCAL NEUROLOGICAL DEFECT		

PRE HOSPITAL TREATMENT

	YES	NO
OXYGEN		
IV FLUIDS		
ANTIBIOTICS		
ANTIVIRALS		

AT PICU ADMISSION:

	YES	NO
AVPU SCALE		
GCS SCORE		
ABC		
ICT		
SEIZURE		

INVESTIGATIONS:

CBC: Hb TC DC PLATELET ESR

RFT:

UREA

CREATINE

SERUM ELECTROLYTES:

LFT:

CSF:

APPEARANCE

CELL COUNT

PROTEIN

SUGAR

VIRAL MARKERS

GENE EXPERT

IMAGING:

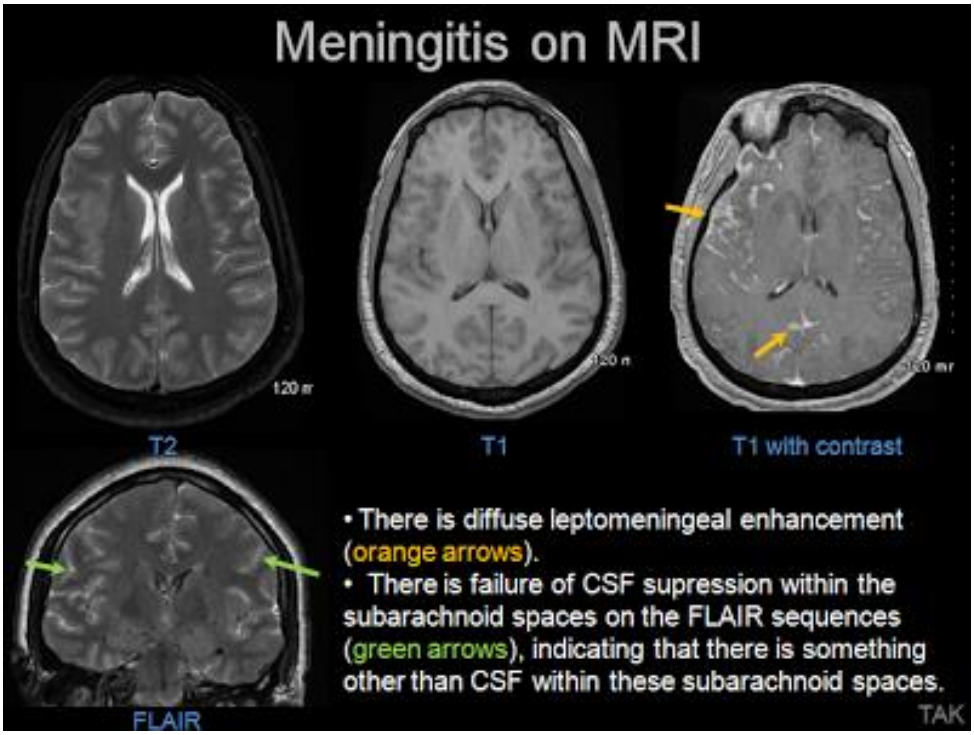
MRI

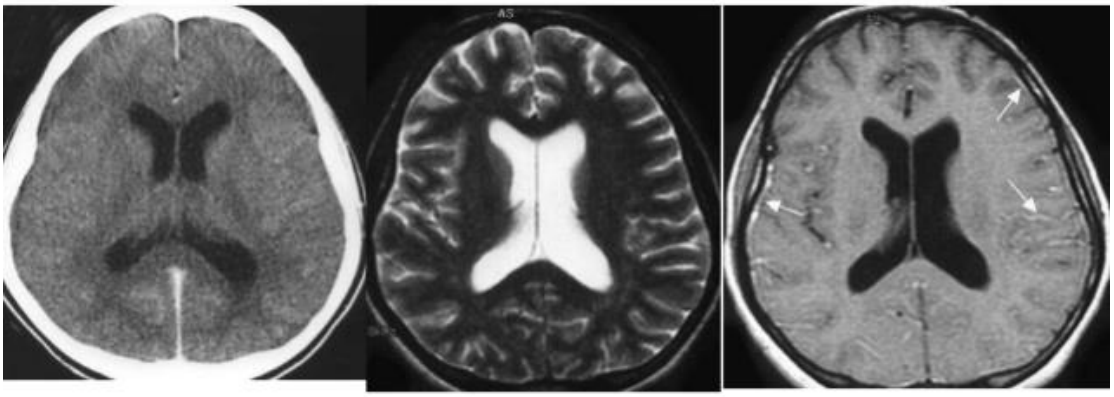
CT

EEG

OUTCOME : DEATH/ALIVE

IF ALIVE, SEQUELAE IF ANY



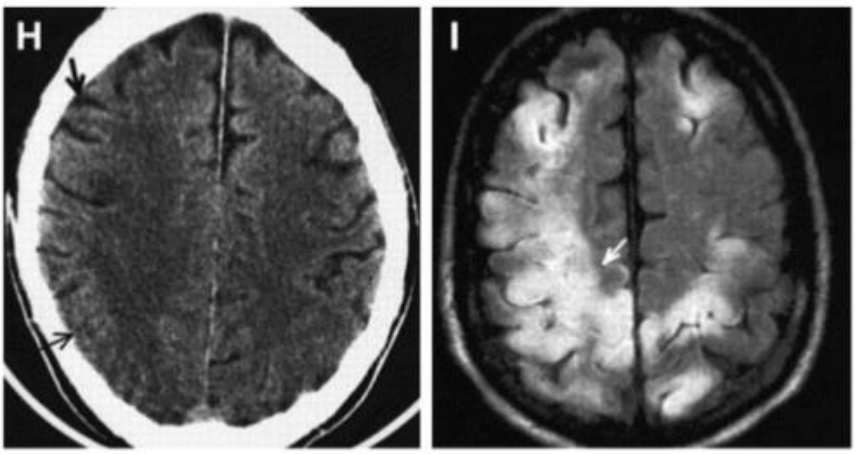


This axial nonenhanced computed tomography scan shows mild ventriculomegaly and sulcal effacement

Acute bacterial meningitis. This axial T2-weighted magnetic resonance image shows only mild ventriculomegaly.

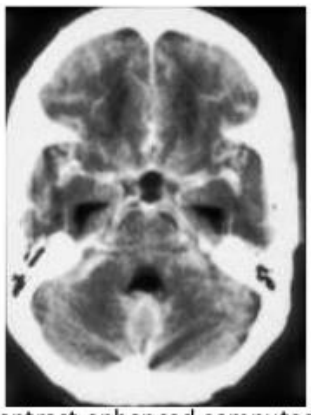
This contrast-enhanced, axial T1-weighted magnetic resonance image shows leptomeningeal enhancement (*arrows*).

Viral Encephalitis

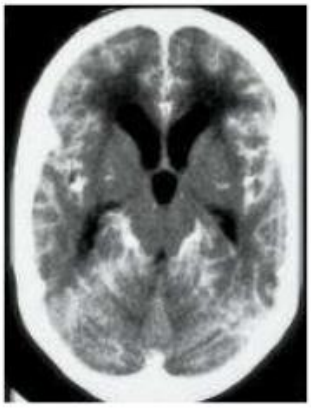


- Brain imaging is frequently normal in viral encephalitis. Occasionally, nonspecific changes consist of either sulcal effacement (H) (thin arrow), compared with normal sulcal spaces (thick arrow); or increased signal (I) (arrow), reflecting increased water content in the mildly swollen brain of the same patient
- These changes developed in a patient with probable enterovirus encephalitis but can be produced by many viruses, as well as after head injury and in various metabolic encephalopathies.

TB Meningitis



Contrast-enhanced computed tomography (CT) scan in a patient with tuberculous meningitis demonstrating marked enhancement in the basal cistern and meninges, with dilatation of the ventricles.



Contrast-enhanced computed tomography (CT) scan of a child with tuberculous meningitis demonstrating acute hydrocephalus and meningeal enhancement.

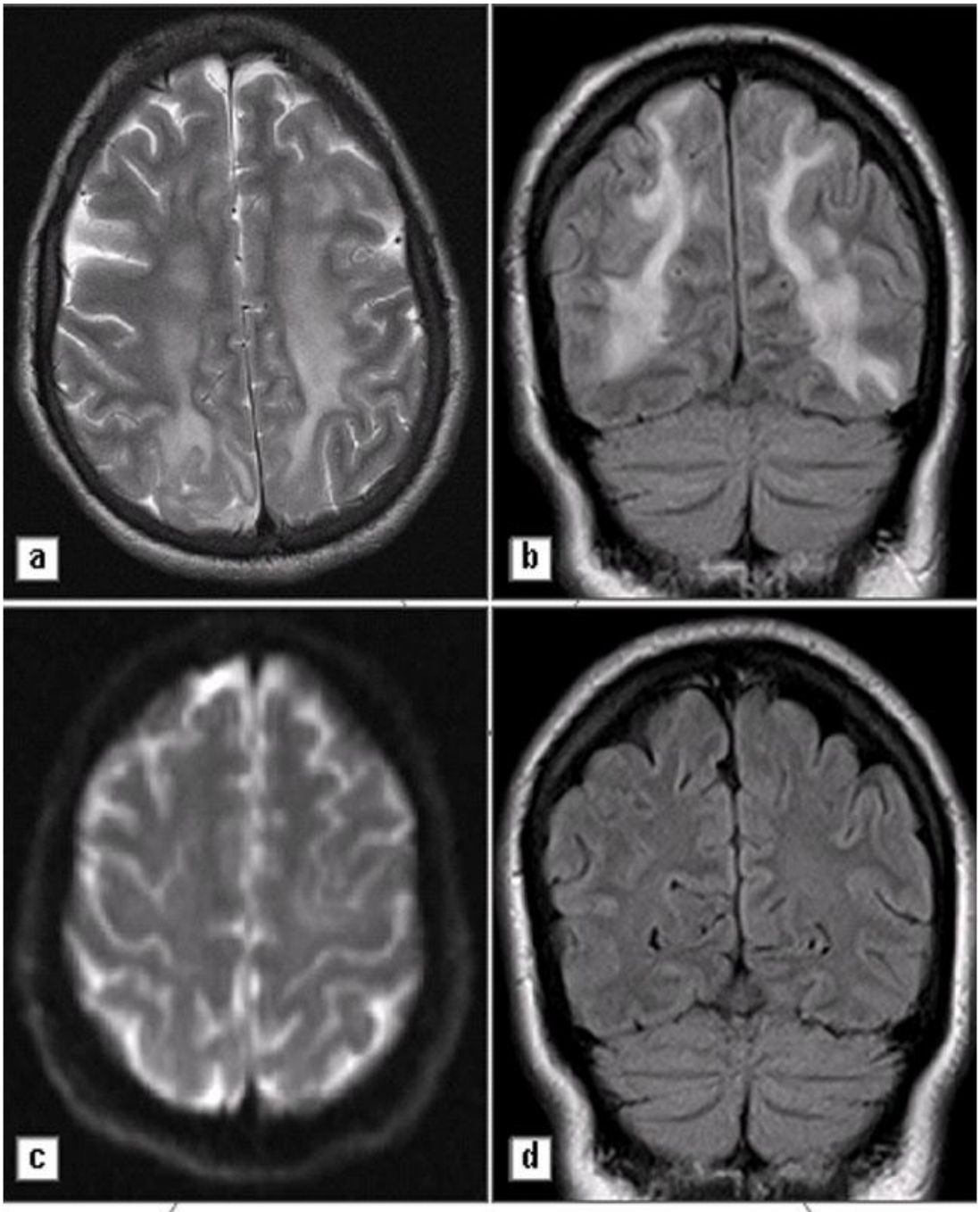
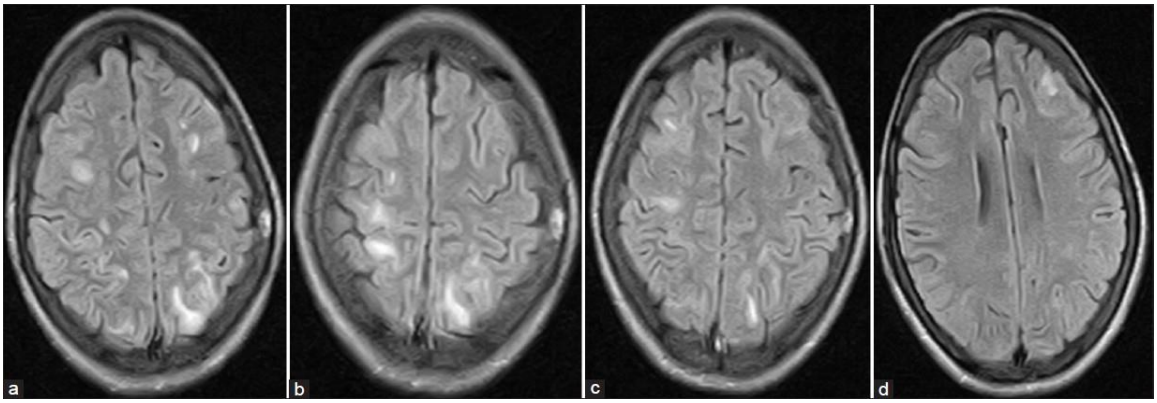


Figure-1: (a) T2-weighted axial image shows diffuse hyperintense signal in deep white matter and subcortical white matter in bilateral frontal and occipital regions. (b) Coronal FLAIR image shows diffuse hyperintense signal in deep white matter and subcortical white matter along the frontal and occipital regions. (c) Diffusion weighted image shows no evidence of diffusion restriction. (d) Follow-up Coronal FLAIR image shows complete resolution of previously noted abnormal signals.


a
b
c
d