Parenchymal CNS infections

Dr. Kifah alubaidy

Parenchymal CNS infections

Infection of the substance of the nervous system depending on the acuteness of the infection and the type of organism will produce symptoms of

- focal deficits and/or seizures
- general signs of infection

- Viral
  - Encephalitis
  - Rabies
  - Subacute sclerosing panencephalitis
  - Progressive multifocal leucoencephalopathy
  - Poliomyelitis
- Bacterial
  - Cerebral abscess
  - Subdural empyema
  - Spinal epidural abscess
  - Lyme disease
  - Neurosyphilis

Viral encephalitis

The term “acute viral encephalitis” is used to describe restricted CNS involvement however, most CNS viral infections causing mild meningoencephalitis rather than pure encephalitis.

In Europe, the most serious cause of viral encephalitis is herpes simplex which probably reaches the brain via the olfactory nerves. Varicella zoster is also an important cause.

viruses can be transmitted by mosquitoes and ticks (arboviruses) Japanese encephalitis & West Nile virus

HIV may cause encephalitis with a subacute or chronic presentation

Viral encephalitis

Pathophysiology

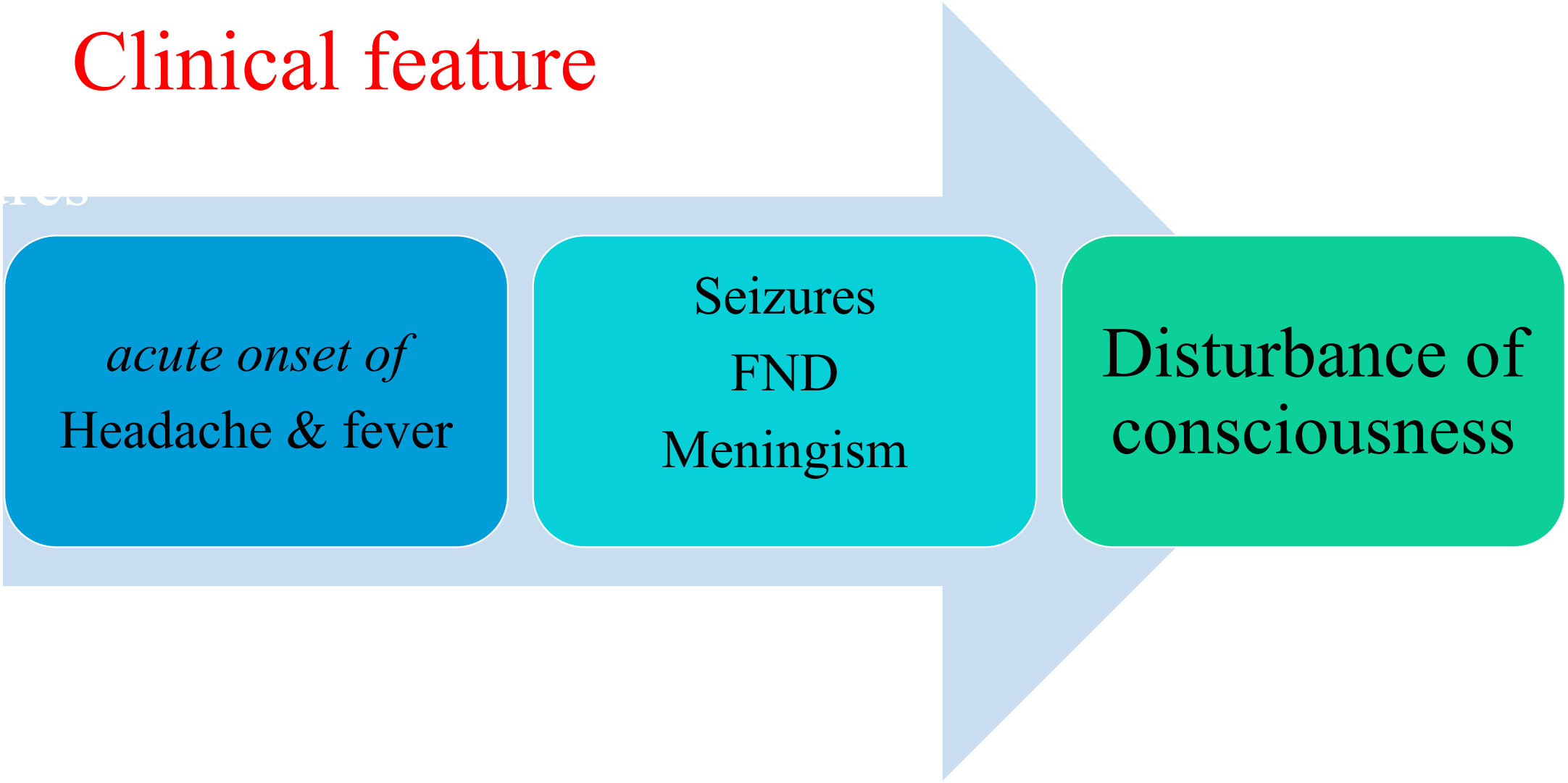
The infection provokes an inflammatory response that involves the cortex, white matter, basal ganglia and brainstem.

The perivascular space of brain is infiltrated by polymorphonuclear cells with neuronal degeneration and diffuse glial proliferation, often associated with cerebral edema.

The distribution of lesions varies with the type of virus.

Herpes simplex encephalitis	**temporal lobes**
Cytomegalovirus	areas adjacent to the ventricles
West Nile virus	basal ganglia & thalamus

Clinical feature



Specific Clinical Patterns

Virus (Family)	Specific Clinical Patterns	Season
HSV	subacute psychiatric manifestation, recurrent meningitis	All year
VZV	Rash	Late winter, spring
Influenza virus	Reversible frontal syndrome, myelitis	winter

Specific Clinical Patterns

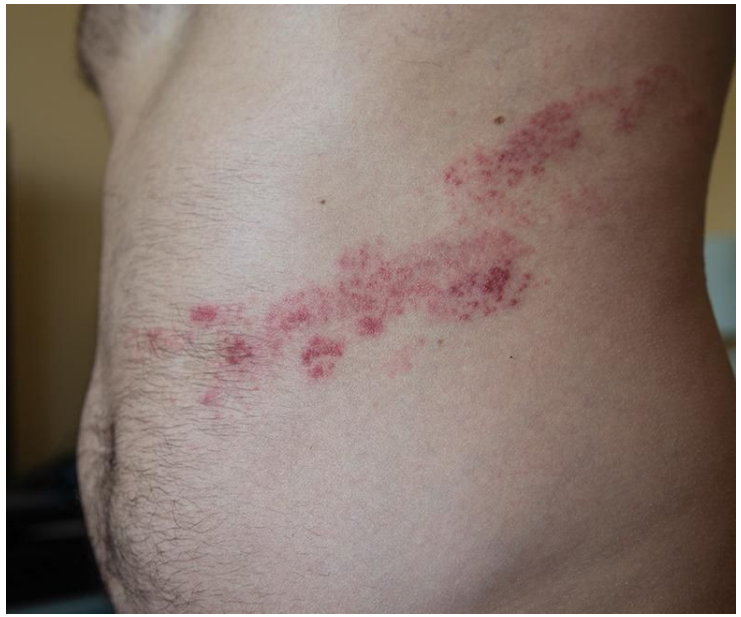
Virus (Family)	Specific Clinical Patterns	Season
Enteroviruses	Herpangina; hand, foot, mouth disease	Summer
Rabies virus	Paresthesias; confusion, spasms, hydrophobia; brainstem features	Dogs bit
Mumps virus	Parotitis, pancreatitis, orchitis, aseptic meningitis	Winter and spring

Specific Clinical Patterns

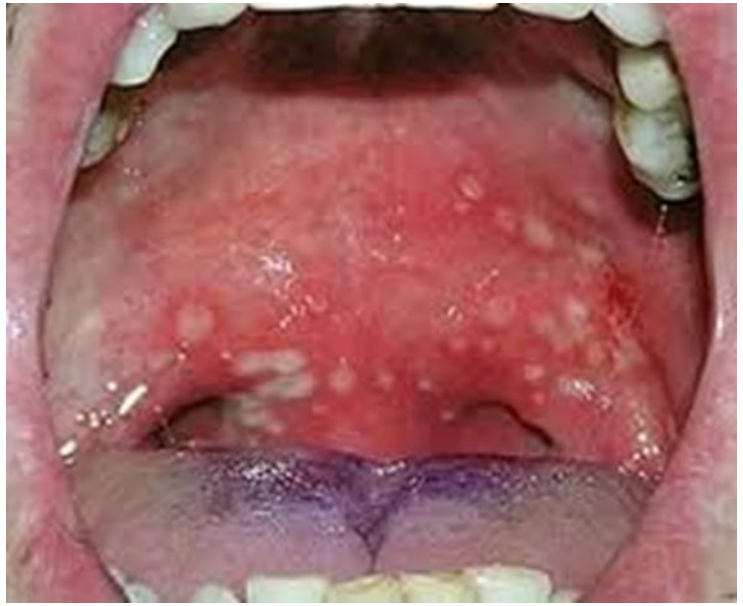
Virus (Family)	Specific Clinical Patterns	Season
Measles virus	Characteristic rash; myelitis	Winter and spring
Lymphocytic choriomeningitis virus	orchitis; leukopenia, thrombocytopenia	winter
West Nile virus	Motor or brainstem involvement	Summer

Rash

VZV rash



Herpangina



Investigations

Blood and Skin Cultures

All patients with encephalitis should have blood cultures to rule out bacterial and fungal infections.

Serologic Tests

serum IgM antibodies (ELISAs) detecting varicella and arboviruses.

A PCR-based test for rapid detection of West Nile virus

Investigations

CSF examination

To establish the diagnosis and reveals typical viral profile:

- Protein: mildly to moderately elevated (60-80 mg/dL)
- Glucose: normal
- WBC : moderate pleocytosis (up to 1000 leukocytes/µL)

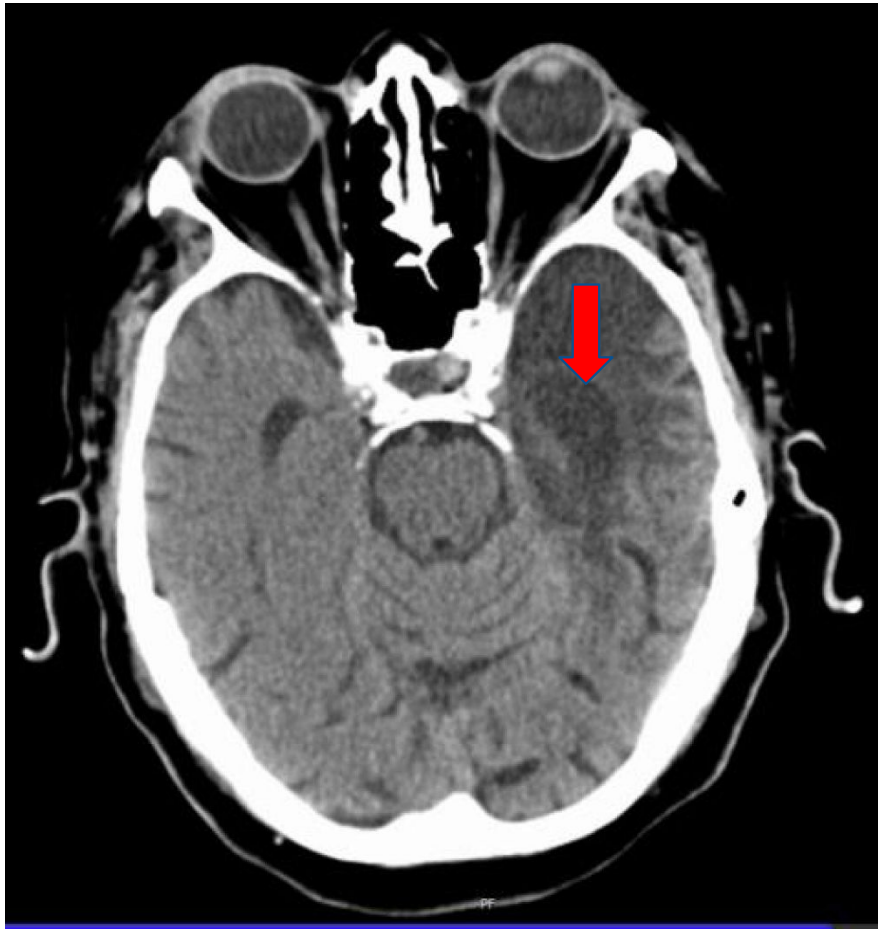
  Type; Mononuclear cells usually predominate
- PCR evaluation to detect herpes simplex virus (HSV) DNA

Up to 10% of the patients with viral encephalitis may have completely normal CSF studies.

Immiging

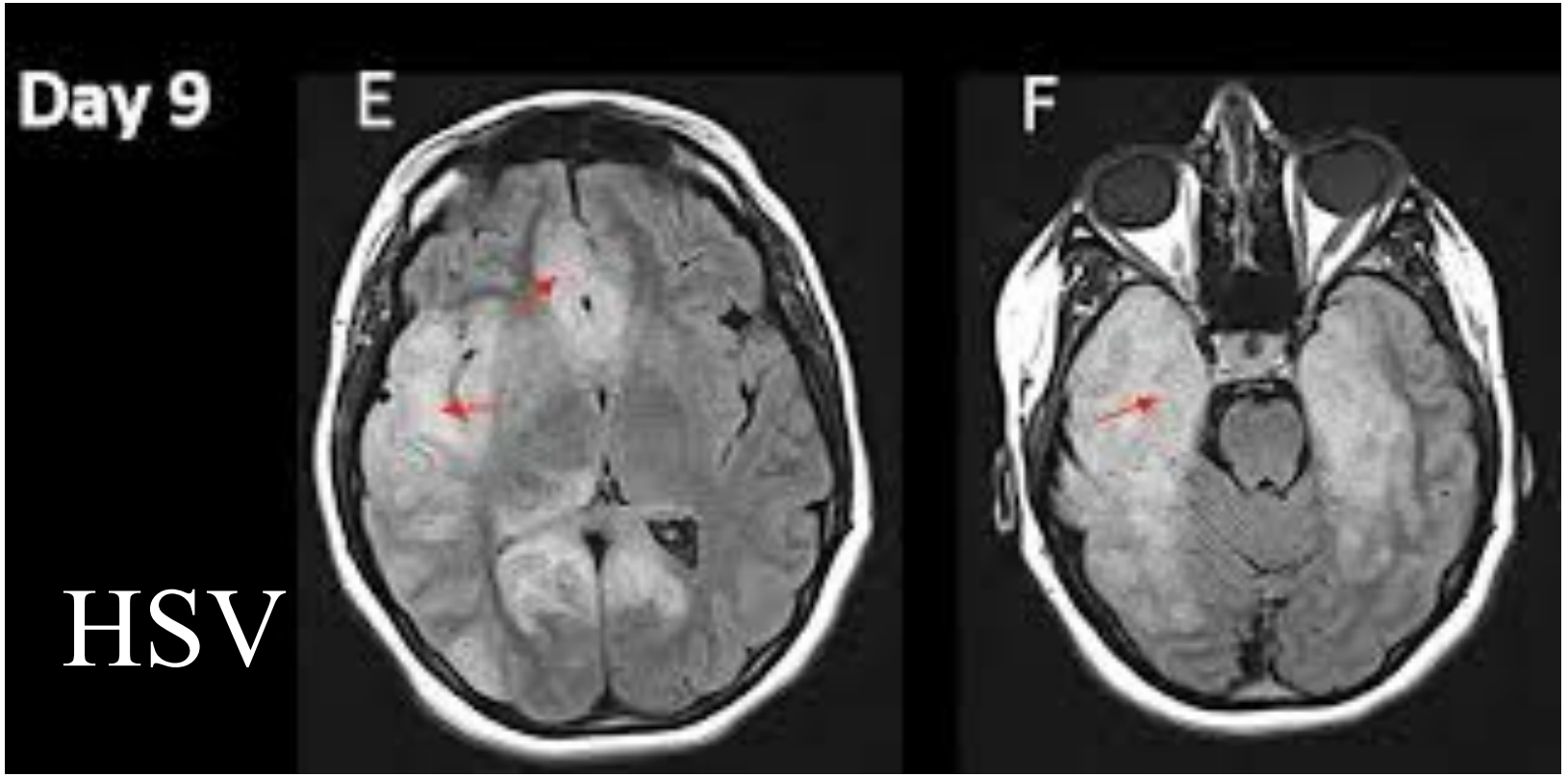
CT scanning

In HSV encephalitis, may show low-density lesions in the temporal lobes, which may not be present until 3-4 days after onset. Edema and hemorrhages may be found, and, after 1 week, contrast enhancement may be observed.



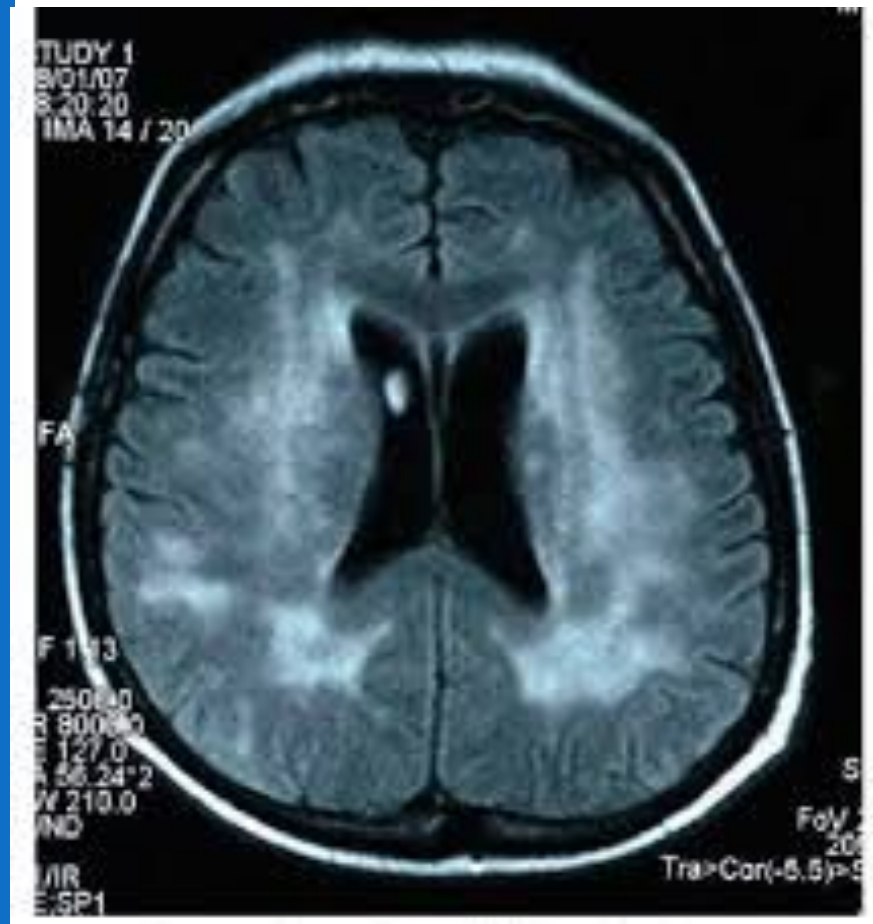
Investigations

MRI is more sensitive and specific than CT for identifying viral encephalitis, especially in the early phase

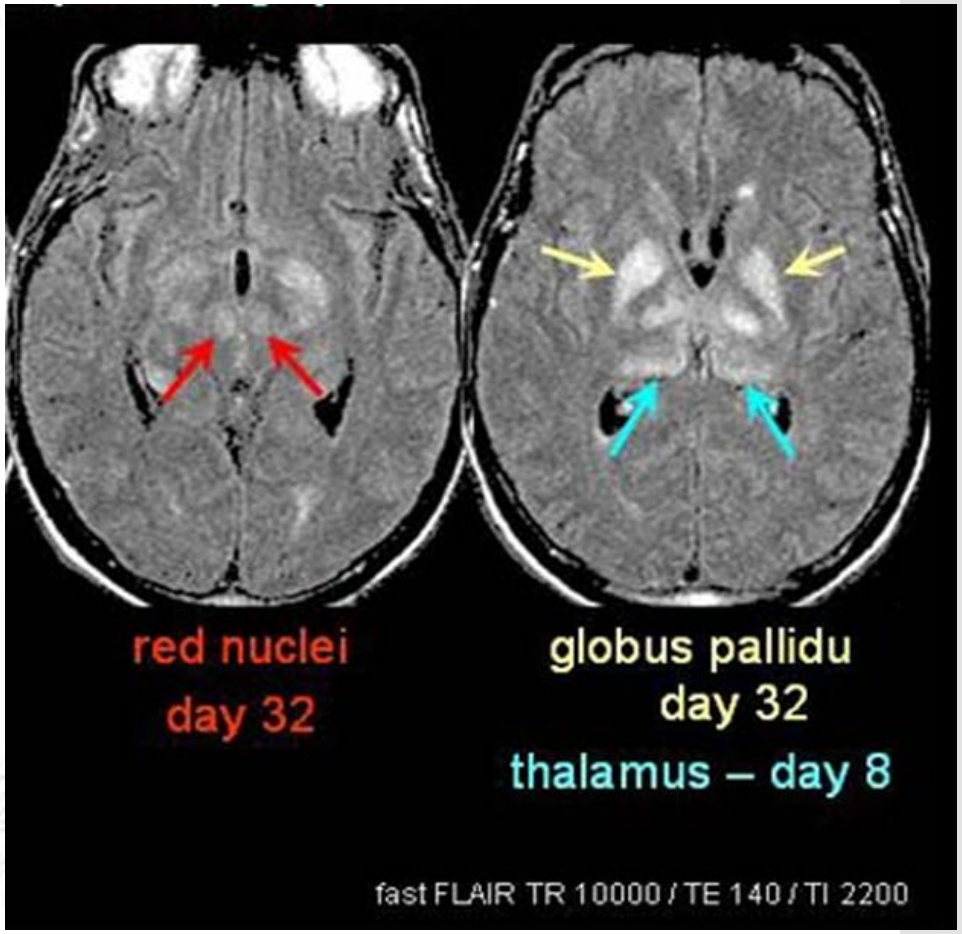


Investigations MRI

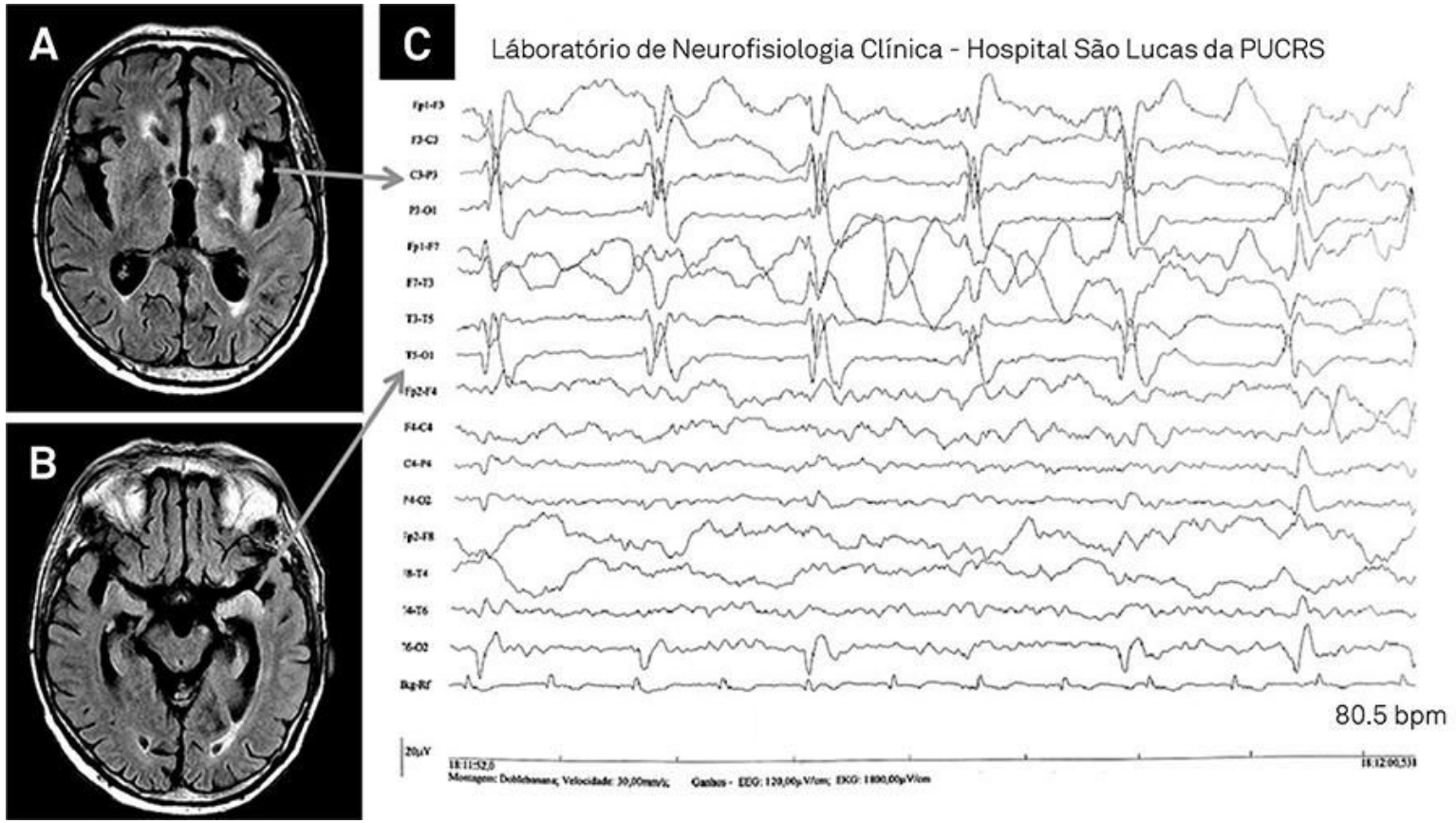
Cytomegalovirus



West Nile virus



EEG is usually abnormal in the early stages, especially in herpes simplex encephalitis, with characteristic periodic slow wave activity in the temporal lobes



Treatment

Medical care should be devoted to appropriate management of the airway, bladder function, fluid and electrolyte balance, nutrition, prevention of bedsores, secondary pulmonary infection, and hyperpyrexia.

Optimum treatment for herpes simplex encephalitis (acyclovir 10 mg/kg IV 3 times daily for 2–3 weeks) has reduced mortality from 70% to around 10%. This should be given early to all patients suspected of having viral encephalitis

Increased ICP should be managed in the ICU setting with head elevation, gentle diuresis, mannitol, and hyperventilation

Seizure activity should control by IV Phenytoin and valproic acid

The use of corticosteroids as an adjunctive therapy for viral encephalitis is controversial and currently being evaluated

Delayed diagnosis of herpes simplex encephalitis (HSE) increases morbidity and mortality rates

Brainstem encephalitis

Presentation

with ataxia, dysarthria, diplopia or other cranial nerve palsies.

Causative agent

is presumed to be viral. However, Listeria monocytogenes may cause a similar syndrome with meningitis

Treatment

ampicillin 500 mg 4 times daily

Poliomyelitis

Pathophysiology

is caused by one of three polioviruses, Infection usually occurs through the nasopharynx. The virus causes a lymphocytic meningitis and infects the grey matter of the spinal cord, brainstem and cortex. There is a particular propensity to damage anterior horn cells, especially in the lumbar segments.

Clinical features

The incubation period is 7–14 days. the initial phase of a few days of mild fever and headache, patients recover fully. In some individuals, after a week of well-being, there is a recurrence of pyrexia, headache and meningism.

Weakness may start later in one muscle group and can progress to widespread paresis to respiratory failure. gradual recovery may then take place over several months

Management

bed rest, respiratory difficulties require tracheostomy and ventilation. Subsequent treatment is by physiotherapy and orthopaedic measures.

Prevention

immunization with live (Sabin) vaccine.

Subacute sclerosing panencephalitis

- It is a rare, chronic, progressive and eventually fatal complication of measles, resulting from inability of the nervous system to eradicate the virus.
- It occurs in children and adolescents, usually many years after the primary virus infection.
- Started as insidious onset of intellectual deterioration, apathy and clumsiness, followed by myoclonic jerks, rigidity and dementia.
- The CSF may show a mild lymphocytic pleocytosis
- EEG demonstrates characteristic periodic bursts of triphasic waves.
- Although there is persistent measles-specific IgG in serum and CSF, antiviral therapy is ineffective and death ensues within a few years.

Progressive multifocal leucoencephal opathy

- It is an infection of oligodendrocytes by human polyomavirus JC, causing widespread demyelination of the white matter of the cerebral hemispheres.

It is a rare complication of

- Lymphoma
- leukemia
- carcinomatosis
- AIDS
- secondary to immunosuppression
  - organ transplantation
  - disease-modifying drugs for MS.

Progressive multifocal leukoencephal opathy

Clinical signs

- rapidly progressive Dementia
- hemiparesis and aphasia

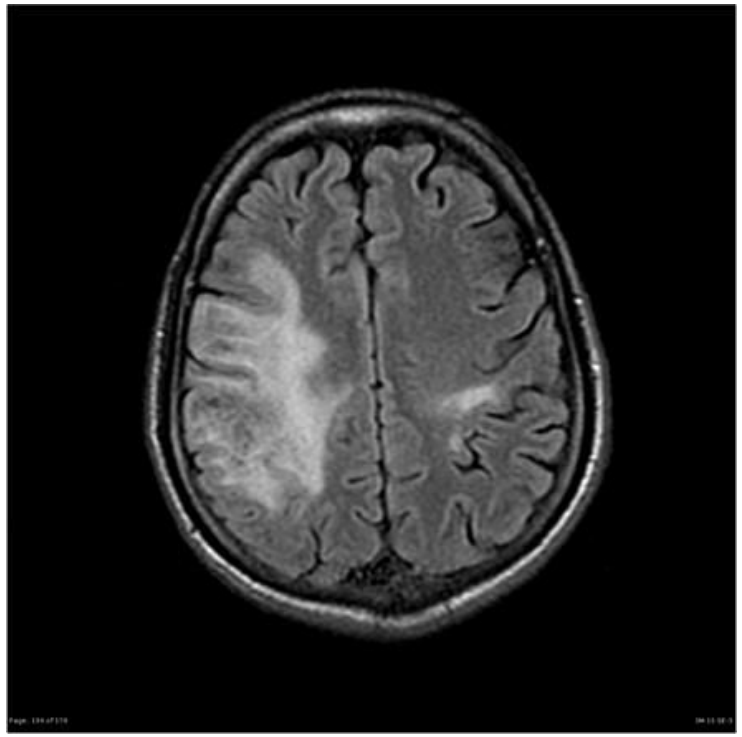
usually leading to death within weeks or months

MRI

diffuse high signal in the cerebral white matter on T2-weighted images.

Treatment

is restoration of the immune response (by treating AIDS or reversing immunosuppression)



Cerebral abscess

Causes

- penetrating injury
- spread from paranasal sinuses or middle ear
- Haematogenous spread (congenital heart disease) *may lead to multiple abscesses*.

- The causative organism & site the of abscess are both related to the source of infection

Cerebral abscess etiology

Site of abscess	Source of infection	Likely organisms
Frontal lobe	Paranasal sinuses & Teeth	Streptococci Anaerobes
Temporal lobe	Middle ear	Streptococci Enterobacteriaceae
Cerebellum	Sphenoid sinus Mastoid/middle ear	Pseudomonas spp. Anaerobes
Any site	Penetrating trauma	Staphylococci
Multiple	Metastatic and cryptogenic endocarditis or cyanotic heart disease	Streptococci Anaerobes

Cerebral abscess

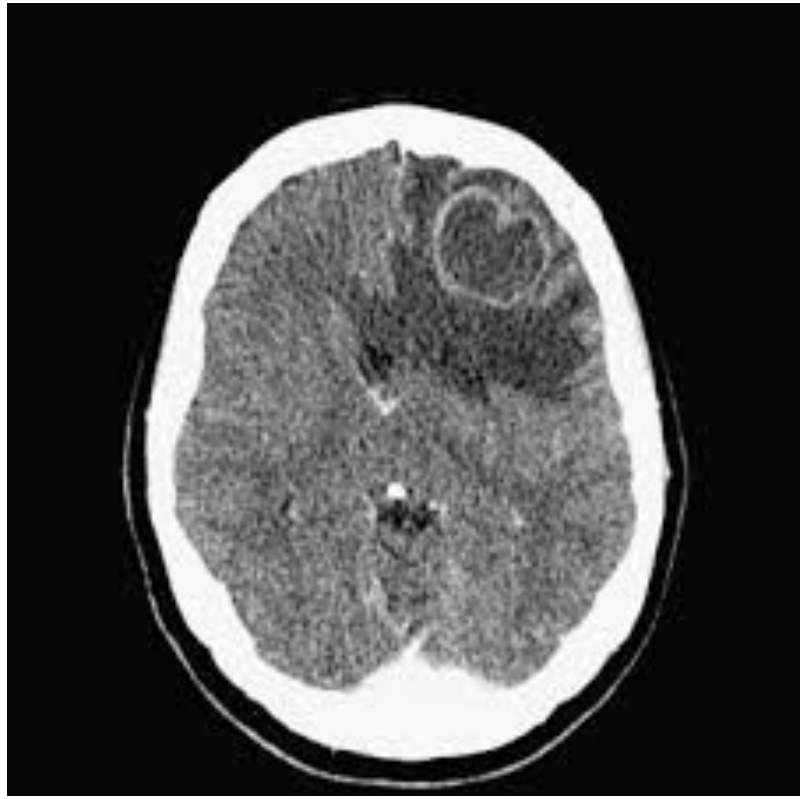
Clinical features

- acutely
  - Fever
  - Headache
  - meningism
  - drowsiness
- Subacute (commonly) presents over days or weeks as a cerebral mass
  - focal hemisphere signs
  - Seizures
  - raised intracranial pressure

Investigations

- There may be an elevated white blood cell count and ESR.
- Lumbar puncture is potentially hazardous

CT reveals single or multiple low-density areas, which show ring enhancement with contrast and surrounding cerebral edema



Management and prognosis

- Antimicrobial therapy is indicated once the diagnosis is made. The likely source of infection should guide the choice of antibiotic.
- In general either Cefotaxime 2–3 g IV 4 times daily or Ceftazidime 2 g IV 3 times daily plus Metronidazole 500 mg IV 3 times daily
- In Cerebellum add Gentamicin* 5 mg/kg IV daily
- Neurosurgical patients or Penetrating trauma add Flucloxacillin 2–3 g IV 4 times daily or vancomycin should be considered.
- Surgical drainage by burr-hole aspiration or excision may be necessary, especially where the presence of a capsule may lead to a persistent focus of infection.
- Epilepsy frequently develops and is often resistant to treatment.
- Despite advances in therapy, mortality remains 10–20% and may partly relate to delay in diagnosis and treatment