VIRAL MENINGOENCEPHALITIS

:Lecturer

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:Objectives

- Etiology of viral meningoencephalitis.
- Pathology of viral meningoencephalitis.
- Clinical manifestations of viral meningoencephalitis.
- Invstigations of viral meningoencephalitis.
- Treatment of viral meningoencephalitis.
- Prognosis of viral meningoencephalitis.
- Prevention of viral meningoencephalitis.

Viral meningoencephalitis is the most common .cause of CNS infection

:Etiology

Enteroviruses are the most common cause of viral· meningoencephalitis. It spread directly from person to person with incubation period of ≈ 5 days and the disease ranges from mild, self-limited illness to severe encephalitis resulting in death or .significant sequelae

- Herpes simplex virus type 1 (HSV-1) is an important cause of severe, sporadic encephalitis. Brain involvement usually is focal with progression to coma and death in many cases without antiviral therapy.
- Herpes simplex virus type 2 (HSV-2) may cause severe encephalitis with diffuse brain involvement in neonates who usually contract the virus from their mothers at delivery.
- Varicella-zoster virus (VZV) may cause CNS infection (especially cerebellar ataxia) in associated with chickenpox.

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- Cytomegalovirus (CMV) infection of the CNS may be part of congenital infection or disseminated disease in immunocompromised hosts, but infection does not occur in normal infants and children.
- Epstein-Barr virus (EBV) has been associated with myriad (numerous) CNS syndromes e.g. Alice in Wonderland syndrome.

Measles virus can cause acute, subacute or· .subacute sclerosing parencephalitis (SSPE)

Mumps virus meningoencephalitis is mild, however · .deafness may occur

Rabies virus cause the most severe encephalitis that· .is always fatal

Arboviruses are arthropod-borne agents,· .responsible for some cases during summer months

West Nile virus is common in some areas of the· .world

Meningoencephalitis may occasionally caused by · respiratory viruses or it may follow live virus .vaccins

:Pathology

Neurologic damage is caused by direct invasion and destruction of neural tissues by actively multiplying viruses or by host reaction to viral antigens. A marked degree of demyelination with preservation of neurons and their axons represent “postinfectious” .or “allergic” encephalitis

Most viruses cause diffuse encephalitis; whereas HSV-1 cause severe focal encephalitis in the temporal .lobe. Rabies virus is mainly affect the basal ganglia

:Clinical manifestations

The clinical course resulting from infection with the same pathogen is widely variable. Some children may appear initially mildly affected, only to lapse into coma and die suddenly. In others, the illness .may be very severe followed by complete recovery

The onset of illness is generally acute. The presenting manifestations in older children are headache & hyperesthesia; whereas in infants, .irritability & lethargy

Other manifestations include: fever, nausea, vomiting, photophobia, pain in neck, back and legs, mental dullness progressing to stupor in combination with bizarre movements and .convulsions

There also may be focal neurologic signs, unprovoked emotional outbursts & loss of bowel .and bladder control

Enteroviruses may cause anterior horn cell injury and acute flaccid paralysis. Exanthemas (skin rash) may precede or accompany the CNS signs, especially with echoviruses, coxsackieviruses, VZV, .measles, rubella

:Investigations

.CSF finding; see the table above·

Serology of blood may be useful in determining the· etiology of some viral CNS infection e.g. arboviral .infection, but it is of no value in Enteroviruses

.EEG show diffuse slow wave activity·

.CT & MRI show swelling of brain parenchyma ·

Note: HSV encephalitis is suggested by focal seizures & focal finding on EEG, CT or MRI especially if .involve the temporal lobe

:Treatment

HSV encephalitis is treated with IV Acyclovir, 10 mg/kg every 8 hr by infusion over 1 hr for 14–21 days. Otherwise, Rx of viral meningoencephalitis is supportive (although some literatures suggest .Pleconril for enteroviral infection)

Mild disease may require only symptomatic relief. More severe disease may require hospitalization .and intensive care

- Headache and hyperesthesia are treated with rest, non–aspirincontaining analgesics & reduction in .room light, noise, and visitors
- Pain may require codeine, morphine (but better .avoided)
- .Fever; acetaminophen
- Poor oral intake; IV fluids same of that in bacterial meningitis. TPN may be required in prolonged .coma

- Anticipate and be prepared to manage; convulsions, cerebral edema, inadequate respiratory exchange, ARDS, disturbed fluid and electrolyte balance (commonly due to SIADH), aspiration and asphyxia, cardiac or respiratory arrest of central origin.
- ICP should be closely monitored (better by ↑ pressure transducer in the epidural space) & managed accordingly.

Prognosis:

Most children recover completely from viral infections of the CNS, especially those due to enteroviruses, whereas others have high mortality rate e.g. rabies & HSV, or have severe sequelae e.g. intellectual, motor, psychiatric, epileptic, visual, or auditory. Therefore, neurodevelopmental and audiologic evaluations should be part of the .routine follow-up

:Prevention

Isolation of cases, vaccination is available for some viruses e.g. rabies, varicella, & measles with control .of vector for some viral infections e.g. arboviruses