Meningococcal meningoencephalitis

- Community-Acquired Bacterial Meningoencephalitis: The New Guideline
- Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) Initially Presenting as Septic Meningoencephalitis in a 16-Year-Old Male
- Paediatric Active Enhanced Disease Surveillance (PAEDS) annual report 2016: Prospective hospital-based surveillance for serious paediatric conditions
- Virulence Traits of a Serogroup C Meningococcus and Isogenic cssA Mutant, Defective in Surface-Exposed Sialic Acid, in a Murine Model of Meningitis
- EGYPTIAN EOSINOPHILIC AND INFECTIOUS MENINGOENCEPHA- LITIS AND THEIR IMPACT ON PSYCHOLOGICAL ASPECTS
- Invasive meningococcal disease in England: assessing disease burden through linkage of multiple national data sources
- Acute care of patients with bacterial meningitis
- Retinal haemorrhages associated with fatal paediatric infections

Meningococcal meningoencephalitis is a severe and potentially life-threatening condition caused by the bacterium Neisseria meningitidis, also known as meningococcus. Neisseria meningitidis is a leading cause of bacterial meningitis, which is inflammation of the membranes surrounding the brain and spinal cord.

Here are some key points about meningococcal meningoencephalitis

Causative Agent

The condition is caused by the bacterium Neisseria meningitidis, specifically the serogroups A, B, C, W, X, and Y.

Transmission

Meningococcal bacteria are typically transmitted through respiratory droplets from infected individuals. Close contact with an infected person, such as through coughing or kissing, can lead to the spread of the bacteria.

Clinical Presentation

Meningococcal meningoencephalitis presents with symptoms of meningitis, such as severe headache, fever, stiff neck, and photophobia. In addition to these, encephalitis symptoms may include confusion, altered mental status, seizures, and focal neurological deficits.

Diagnosis

Diagnosis involves clinical evaluation, a lumbar puncture to analyze cerebrospinal fluid, and laboratory tests to identify the bacteria. Imaging studies like CT scans or MRIs may also be performed to assess brain involvement.

Treatment

Prompt medical intervention is crucial. Treatment often involves intravenous antibiotics, such as ceftriaxone or cefotaxime. Supportive care, including measures to reduce intracranial pressure, may also be necessary.

Complications

Meningococcal infections can lead to severe complications, including septicemia (bloodstream infection), shock, and organ failure. Neurological complications, such as cerebral edema and hemorrhage, can occur in meningococcal meningoencephalitis.

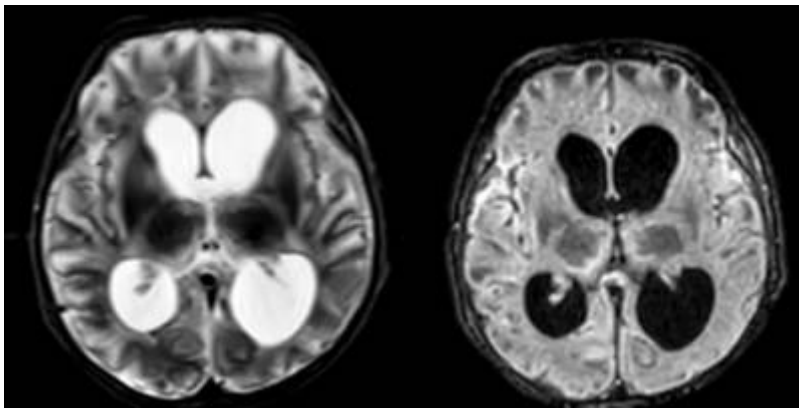
Prevention

Vaccination is a key preventive measure. Meningococcal vaccines are available and are recommended, especially for adolescents, college students, and individuals in certain high-risk groups.

Because meningococcal meningoencephalitis can progress rapidly and have serious consequences, early recognition of symptoms, prompt medical attention, and preventive measures are crucial in managing and preventing the disease.

Case report from the HGUA

A 25-day-old neonate presents to the Pediatric Emergency Department (UPED) with febrile syndrome and possible disconnection from the surroundings. The parents report a slight fever during the night that subsided after the administration of oral paracetamol. The infant is exclusively breastfed on demand but has refused the breast this morning. There have been no vomiting episodes, and there's no recent history of respiratory symptoms. This morning, the parents noticed the baby feeling warm and gave oral paracetamol. Following this, they observed an episode of disconnection from the surroundings with hypotonia that spontaneously resolved in less than a minute. There were no tonic-clonic movements. Since this morning, before the episode, the baby has had ptosis of the left eyelid that has not improved.



Diffuse Alteration of Brain Parenchyma, which associates patchy areas of increased signal intensity in the white matter on T2 weighted image and FLAIR sequences with multifocal distribution.

These alternate with patchy areas of signal drop predominantly in the peripheral regions. This is associated with a diffuse alteration of the cerebral cortex showing restricted diffusion and heterogeneous signal, with isolated punctate foci of parenchymal hemorrhage in periventricular locations in the frontal, parietal, and occipital lobes. Additionally, there is peripheral contrast enhancement of both thalami, ventricular wall enhancement, and diffuse enhancement of the meninges. A 3mm subdural collection in the posterior fossa restricts diffusion and presents peripheral enhancement about a subdural empyema.

The findings are consistent with the suspected diagnosis of meningococcal meningoencephalitis, showing signs of cerebritis with cytotoxic and interstitial cerebral edema, punctate hemorrhagic changes, signs of cerebral ischemia, and a subdural collection in the posterior fossa.

Basal cisterns are of normal size and morphology considering the patient's age. No midline structure displacements are observed. The cerebellum and brainstem show no evidence of abnormal signal areas.

The cranial examination reveals no thickness alterations in the bones included in the study or evidence of osteolytic lesions.

The findings are consistent with the suspected diagnosis of meningococcal meningoencephalitis, showing signs of cerebritis with cytotoxic and interstitial cerebral edema, punctate hemorrhagic changes, signs of cerebral ischemia, hydrocephalus, and a subdural collection in the posterior fossa.