BLOCK: Mental health care and Neurology

Neurology section lec.

CNS infection

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Davidson s principles and practice of medicine

Objective

1. What is CNS infection
2. What is meningitis and what is encephalitis?
3. How we can get CSF sample?
4. How we can differentiate between types of meningitis from CSF results?
5. What is impact of early diagnosis and management on survival?

Infections of the nervous system

The clinical features of nervous system infections depend on the location of the infection (the meninges or the parenchyma of the brain and spinal cord), the causative organism (virus, bacterium, fungus or parasite), and whether the infection is acute or chronic.

25.61 Infections of the nervous system

Bacterial infections

- Meningitis
- Suppurative encephalitis
- Brain abscess
- Paravertebral (epidural) abscess
- Tuberculosis (p. 588)
- Neurosyphilis
- Leprosy (Hansen's disease) (peripheral nerves)*
- Diphtheria (peripheral nerves)*
- Tetanus (motor cells)

Viral infections

- Meningitis
- Encephalitis
- Transverse myelitis
- Progressive multifocal leucoencephalopathy
- Poliomyelitis
- Subacute sclerosing panencephalitis (late sequel)
- Rabies
- HIV infection (Ch. 12)

Prion diseases

- Creutzfeldt–Jakob disease
- Kuru

Prion diseases

- Creutzfeldt–Jakob disease
- Kuru

Protozoal infections

- Malaria*
- Toxoplasmosis (in immune-suppressed)*
- Trypanosomiasis*
- Amoebic abscess*

Helminthic infections

- Schistosomiasis (spinal cord)*
- Cysticercosis*
- Hydatid disease*
- Strongyloidiasis*

Fungal infections

- *Candida* meningitis or brain abscess
- Cryptococcal meningitis

Meningitis

Acute infection of the meninges presents with a characteristic combination of pyrexia, headache and meningism. Meningism consists of headache, photophobia and stiffness of the neck, often accompanied by other signs of meningeal irritation, including Kernig's sign (extension at the knee with the hip joint flexed causes spasm in the hamstring muscles) and Brudzinski's sign (passive flexion of the neck causes flexion of the hips and knees).

- Meningism is not specific to meningitis and can occur in patients with subarachnoid haemorrhage. The severity of clinical features varies with the causative organism, as does the presence of other features such as a rash. Abnormalities in the CSF are important in distinguishing the cause of meningitis.

25.6 How to interpret cerebrospinal fluid results

	Normal	Subarachnoid haemorrhage	Acute bacterial meningitis	Viral meningitis	Tuberculous meningitis	Multiple sclerosis
Pressure	50–250 mm of water	Increased	Normal/increased	Normal	Normal/increased	Normal
Colour	Clear	Blood-stained Xanthochromic	Cloudy	Clear	Clear/cloudy	Clear
Red cell count ($\times 10^6$/L)	0–4	Raised	Normal	Normal	Normal	Normal
White cell count ($\times 10^6$/L)	0–4	Normal/slightly raised	1000–5000 polymorphs	10–2000 lymphocytes	50–5000 lymphocytes	0–50 lymphocytes
Glucose	>50–60% of blood level	Normal	Decreased	Normal	Decreased	Normal
Protein	<0.45 g/L	Increased	Increased	Normal/increased	Increased	Normal/increased
Microbiology	Sterile	Sterile	Organisms on Gram stain and/or culture	Sterile/virus detected	Ziehl–Neelsen/auramine stain or tuberculosis culture positive	Sterile
Oligoclonal bands	Negative	Negative	Can be positive	Can be positive	Can be positive	Often positive

Viral meningitis

Viruses are the most common cause of meningitis, usually resulting in a benign and self-limiting illness requiring no specific therapy. It is much less serious than bacterial meningitis unless there is associated encephalitis. A number of viruses can cause meningitis ,the most common being enteroviruses.

Investigations

The diagnosis is made by lumbar puncture. CSF usually contains an **excess of lymphocytes**. While glucose and protein levels are commonly normal, the latter may be raised. It is important to verify that the patient has not received antibiotics (for whatever cause) prior to the lumbar puncture, as CSF lymphocytosis can also be found in partially treated bacterial meningitis.

Management

There is no specific treatment and the condition is usually benign and self-limiting. The patient should be treated symptomatically in a quiet environment. Recovery usually occurs within days, although a lymphocytic pleocytosis may persist in the CSF.

Bacterial meningitis

Many bacteria can cause meningitis but geographical patterns vary, as does age-related sensitivity .Bacterial meningitis is usually part of a bacteremia illness, although direct spread from an adjacent focus of infection in the ear, skull fracture or sinus can be causative. Antibiotics have rendered this less common but mortality and morbidity remain significant. An important factor in determining prognosis is early diagnosis and the prompt initiation of appropriate therapy. The meningococcus and other common causes of meningitis are normal commensals of the upper respiratory tract.

i 25.63 Bacterial causes of meningitis

Age of onset	Common	Less common
Neonate	Gram-negative bacilli (*Escherichia coli, Proteus*) Group B streptococci	*Listeria monocytogenes*
Pre-school child	*Haemophilus influenzae* *Neisseria meningitidis* (subtypes B, C, Y, W) *Streptococcus pneumoniae*	*Mycobacterium tuberculosis*
Older child and adult	*N. meningitidis* (subtypes B, C, Y, W) *Strep. pneumoniae*	*L. monocytogenes* *M. tuberculosis* *Staphylococcus aureus (skull fracture)* *H. influenzae*

Investigations

Lumbar puncture is mandatory unless there are contraindications ;If the patient is drowsy and has focal neurological signs or seizures, is immunosuppressed, has undergone recent neurosurgery or has suffered a head injury, it is wise to obtain a CT to exclude a mass lesion (such as a cerebral abscess) before lumbar puncture because of the risk of coning. This should not, however, delay treatment of presumed meningitis. If lumbar puncture is deferred or omitted, it is essential to take blood cultures and to start empirical treatment. Lumbar puncture will help differentiate the causative organism: in bacterial meningitis the CSF is cloudy (turbid) due to the presence of many neutrophils (often > 1000 × 106 cells/L), the protein content is significantly elevated and the glucose reduced. Gram film and culture may allow identification of the organism. Blood cultures may be positive. PCR techniques can be used on both blood and CSF to identify bacterial DNA.

Management

There is an untreated mortality rate of around 80%, so action must be swift. In suspected bacterial meningitis the patient should be given **parenteral benzylpenicillin immediately** (intravenous is preferable) and prompt hospital admission should be arranged.
Adjunctive **glucocorticoid** therapy is useful in reducing hearing loss and neurological sequelae in both children and adults .In meningococcal disease, mortality is doubled if the patient presents with features of sepsis rather than meningitis. Individuals
likely to require intensive care facilities and expertise include those with cardiac, respiratory or renal involvement, and those with CNS depression prejudicing the airway. Early endotracheal intubation and mechanical ventilation protect the airway and
may prevent the development of the acute respiratory distress
syndrome .Adverse prognostic features include hypotensive shock, a rapidly developing rash, a haemorrhagic diathesis, multisystem failure and age over 60 years.

Prevention of meningococcal infection

Close contacts of patients with meningococcal infection should be given 2 days of oral rifampicin. In adults, a single dose of ciprofloxacin is an alternative. If not treated with ceftriaxone, the index case should be given similar treatment to clear infection from the nasopharynx before hospital discharge.

Vaccines are available for most meningococcal subgroups but not group B, which is one of the most common serogroups isolated in many countries.

Tuberculous meningitis

Tuberculous meningitis is now uncommon in developed countries except in immunocompromised individuals, although it is still seen in those born in endemic areas and in developing countries. It is seen more frequently as a secondary infection in patients with the acquired immunodeficiency syndrome (AIDS).

Pathophysiology

Tuberculous meningitis most commonly occurs shortly after a primary infection in childhood or as part of miliary tuberculosis. The usual local source of infection is a caseous focus in the meninges or brain substance adjacent to the CSF pathway. The brain is covered by a greenish, gelatinous exudate, especially **around the base**, and numerous scattered tubercles are found on the meninges.

Clinical features

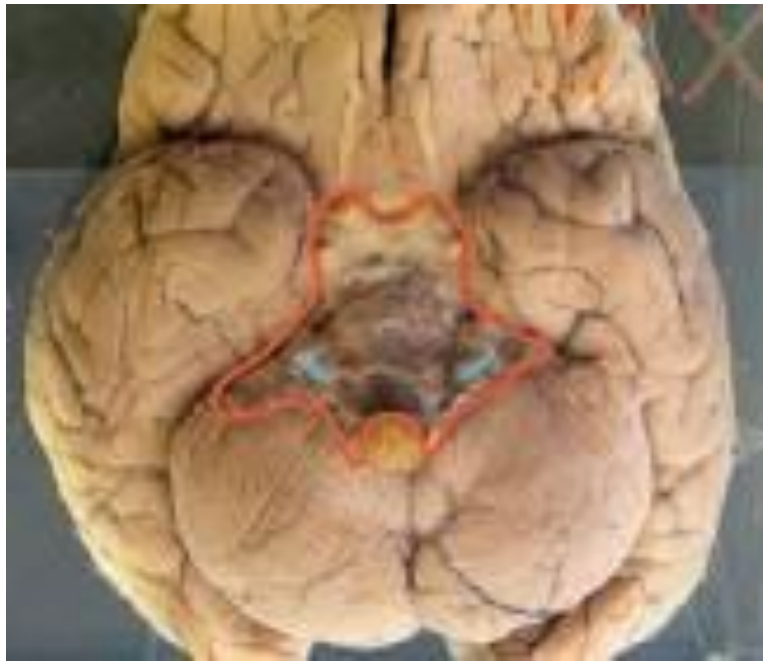
Onset is much slower than in other bacterial meningitis – over 2–8 weeks. If untreated, tuberculous meningitis is fatal in a few weeks but complete recovery is usual if treatment is started early. When treatment is initiated later, the rate of death or serious neurological deficit may be as high as 30%.

Investigations

Lumbar puncture should be performed if the diagnosis is suspected. The CSF is under increased pressure. It is usually clear but, when allowed to stand, a fine clot ('spider web') may form. The fluid contains up to 500 × 106 cells/L, predominantly **lymphocytes**, but can contain neutrophils. There is a rise in protein and a marked fall in glucose. The tubercle bacillus may be detected in a smear of the centrifuged deposit from the CSF but a negative result does not exclude the diagnosis. The CSF should be cultured but, as this result will not be known for up to 6 weeks, treatment must be started without waiting for confirmation. Brain imaging may show hydrocephalus, brisk meningeal enhancement on enhanced CT or MRI, and/or an intracranial tuberculoma.

Management

As soon as the diagnosis is made or strongly suspected, antituberculous therapy should be started. The use of glucocorticoids in addition to antituberculous therapy has been controversial. Recent evidence suggests that it improves mortality, especially if given early, Surgical ventricular drainage may be needed if obstructive hydrocephalus develops. Skilled nursing is essential during the acute phase of the illness, and adequate hydration and nutrition must be maintained.



Parenchymal viral infections

Infection of the substance of the nervous system will produce symptoms of **focal dysfunction** (deficits and/or seizures) **with general signs of infection**, depending on the acuteness of the infection and the type of organism.

Viral encephalitis

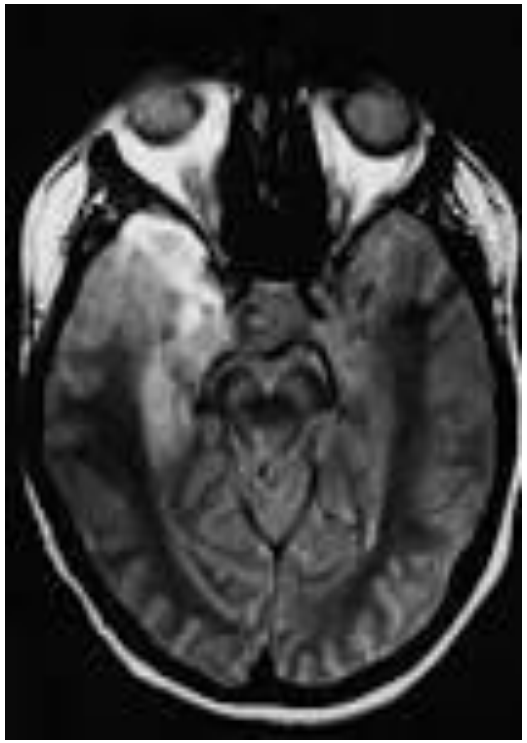
A range of viruses can cause encephalitis but only a minority of patients report recent systemic viral infection. **The most serious cause of viral encephalitis is herpes simplex** which probably reaches the brain via the olfactory nerves. Varicella zoster is also an important cause.

Pathophysiology

The infection provokes an inflammatory response that involves the cortex, white matter, basal ganglia and brainstem. The distribution of lesions varies with the type of virus. For **example, in herpes simplex encephalitis, the temporal lobes are usually primarily affected,** whereas cytomegalovirus can involve the areas adjacent to the ventricles (ventriculitis).

Clinical features

Viral encephalitis presents with acute onset of headache, fever, focal neurological signs (aphasia and/or hemiplegia, visual field defects) and seizures. Disturbance of consciousness ranging from drowsiness to deep coma supervenes early and may advance dramatically.
Meningism occurs in many patients.



Investigations

Imaging by CT scan may show low-density lesions in the temporal lobes but MRI is more sensitive in detecting early abnormalities.

Lumbar puncture should be performed once imaging has excluded a mass lesion. The CSF usually contains **excess lymphocytes** but polymorphonuclear cells may predominate in the early stages.

The CSF may be normal in up to 10% of cases. Some viruses, including the West Nile virus, may cause a sustained neutrophilic CSF. The protein content may be elevated but the glucose is normal. The EEG is usually abnormal in the early stages, especially in herpes simplex encephalitis, with characteristic periodic slow wave activity in the temporal lobes. Virological investigations of the CSF, including PCR, may reveal the causative organism but treatment initiation should not await this.

Management

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.

Some survivors will have residual epilepsy or cognitive impairment.

Antiepileptic treatment may be required and raised intracranial pressure may indicate the need for dexamethasone.

Thanks