CNS INFECTIONS – 3
Brain abscess

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Definition

- A focal, suppurative process within the brain parenchyma
- it begins in an area of devitalized brain tissue as a localized area of cerebritis and develops into a collection of pus surrounded by a well-vascularized capsule.

Epidemiology

- relatively uncommon intracranial infection
- incidence of approx. 1 in 100,000 persons per year
- These days a significant percentage of brain abscesses are not caused by classic pyogenic bacteria, but rather by Toxoplasmi gondii, Aspergillus spp., Nocardia spp., Mycobacteria spp., and fungi such as Cryptococcus neoformans.
- In 20 to 30% of cases no obvious primary source (cryptogenic brain abscess). reflects the importance of brain abscesses in hosts whose immune systems are compromised (HIV infection, organ transplantation, cancer, or immunosuppressive therapy).

Etiology

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  - (1) by direct spread: paranasal sinusitis, otitis media, mastoiditis, or dental infection;
  - (2) following head trauma or a neurosurgical procedure; or
  - (3) as a result of hematogenous spread from a remote site of infection.

  The most common pathogenic agents are Streptococci, Staphylococcus, Bacteriodes, and Proteus and other gram negative bacilli. Actinomyces, Norcardia, and Candida are also found.

Table 1. Etiology of brain abscess according to mode of spread

- **Direct spread**

Pathway of transmission	Location of abscess	Possible etiology of abscess
paranasal sinusitis	frontal lobes	Streptococci, Haemophilus spp., Bacteroides
dental infections	frontal lobes	Streptococci, Prevotella and Bacteroides
otitits media and mastoiditis	temporal, cerebellum	Streptococci, Bacteroides, P. aeruginosa, Enterobacteriaceae

- **hematogenous spread**
  - in areas supplied by the middle cerebral artery (i.e., posterior frontal or parietal lobes)

Pyogenic lung disease	Streptococcus, Actinomyces, Fusobacterium
Infective endocarditis	Viridans streptococci or S. aureus
Urinary sepsis	Enterobacteriaceae or P. aeruginosa
Intraabdominal source	Streptococcus, Enterobacteriaceae, or anaerobes

- Others

Head trauma	S. aureus, Clostridium or Enterobacteriaceae,
Neurosurgical procedure	Staphylococci, Enterobacteriaceae, or P. aeruginosa.

Pathogenesis and histopathology

- the intact brain parenchyma is relatively resistant to infection.
- preexisting or concomitant areas of ischemia, necrosis, or hypoxia in brain tissue
- once bacteria have established infection, brain abscess formation evolves through
  - cerebritis stage (perivascular infiltration of inflammatory cells, with a central core of coagulative necrosis) and
  - capsule formation (well-formed necrotic center surrounded by a dense collagenous capsule).

Clinical presentation

- expanding intracranial mass lesion, rather than as an infectious process
- **Headache**: most common sym, ~ >75% of patients - constant, dull, aching sensation, either hemicranial or generalized, and progressively more severe and refractory to therapy
- **Fever:** only 50% of patients at the time of diagnosis and is typically low-grade
- **Seizure**: new onset of focal or generalized; presenting sign in 25 to 30% of patients
- **Focal neurologic deficit**: initial presentation in >60% of patients.

clinical presentation depends on the location and on the presence of raised ICP, which develops as edema surrounds the evolving abscess

Site/other factors	Signs
frontal lobe abscess	hemiparesis
temporal lobe abscess	Dysphasia/ an upper homonymous quadrantanopia
cerebellar abscess	nystagmus and ataxia
increased ICP	papilledema, nausea and vomiting, and drowsiness or confusion
abscess has ruptured into the ventricle or the infection has spread to the subarachnoid space	meningismus

Diagnosis

- made by neuroimaging studies
- CT has the advantage of greater feasibility in acutely ill patients
- MRI is better for demonstrating abscesses in the early (cerebritis) stages and is superior to CT for identifying abscesses in the posterior fossa
- on a contrast-enhanced CT scan, a mature brain abscess appears as a focal area of hypodensity surrounded by ring enhancement

- microbiologic diagnosis: Gram's stain & c/s of abscess material obtained by stereotactic needle aspiration
- lumbar puncture should not be performed
- EEG may show a high voltage focal slow wave disturbance
- additional laboratory studies: peripheral white blood cell count and ESR

Differential diagnosis

Conditions that can cause headache, fever, focal neurologic signs, and seizure activity include:

- subdural empyema
- bacterial meningitis
- viral meningo-encephalitis
- superior sagittal sinus thrombosis etc

Treatment

- Empirical therapy of a brain abscess depends on the source of infection.
- Typically includes a third-generation cephalosporin (e.g., cefotaxime) and metronidazole.

Table 2. Empirical Therapy

Source	Antimicrobial Therapy
Paranasal sinusitis	Penicillin or a third-generation cephalosporin + metronidazole
Otitis media	Penicillin + metronidazole + ceftazidime
Dental infection	Penicillin + metronidazole (500 iv TDS
Endocarditis	Nafcillin or vancomycin + metronidazole + third-generation cephalosporin
Hematogenous spread	Ceftazidime + metronidazole + penicillin (3 lacks units/kg/d; max 24 million units (in divided dose 4th hourly)
Head trauma	Nafcillin (2g Q6h) or vancomycin + third-generation cephalosporin
Neurosurgical procedure	Vancomycin 1 gm Q12h + ceftazidime 2gm TDS

- Nafcillin/ vancomycin : multiple abscesses coz of hematogenous spread, or abscesses following head trauma, for coverage of staphylococci.
- Abscesses following neurosurgical procedures: Rx Vancomycin plus ceftazidime for coverage of both staphylococci and P. aeruginosa.

- Aspiration and drainage of the abscess under stereotactic guidance: diagnosis and therapeutic: decompress large abscesses or abscesses that block or threaten to rupture into ventricular system.
- Empirical antibiotic coverage can be modified based on Gram's stain and culture.
- Repeated aspirations may be necessary to control a mass effect.
- Complete excision of a bacterial abscess via craniotomy or craniectomy is generally reserved for multiloculated abscesses or those in which stereotactic aspiration is unsuccessful.
- All patients should receive a minimum of 6 to 8 weeks of parenteral antibiotic therapy.

Anticonvulsant therapy

- Prophylactic anticonvulsant therapy: In addition to surgical drainage and antibiotic therapy, Rx
- continued for at least 3 months after resolution of the abscess, and decisions regarding withdrawal are then based on the EEG.
- If the EEG is abnormal, anticonvulsant therapy should be continued. If the EEG is normal, anticonvulsant therapy can be slowly withdrawn.

- Glucocorticoids: not routinely used, unless substantial peri-abscess edema and mass effect and increased ICP. IV dexamethasone (10 mg every 6 h)
- Serial CT or MRI scans: to document resolution of the abscess. monthly or twice-monthly basis.
- A small amount of enhancement may remain for months after the abscess has been successfully treated.

Prognosis

- Bacterial abscess can be successfully treated in the majority of patients
- Seizures, however, are a common complication and occur in as many as 70% of patients.